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SOME OBSERVATIONS ON THE OCCURRENCE OF AND MODIFICATIONS IN THE TYPE OF VESTIBULAR NYSTAGMUS AFTER EXPERIMENTAL LESION OF THE CENTRAL NERVOUS SYSTEM.*†‡

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An earlier excursion of one of us[1] into the confused and baffling field of the mechanism of vestibular nystagmus left him with a feeling of the futility of attempts to carry the field by direct assault. There were too many perplexing questions on the nature of functional organization of nervous mechanisms to make such an attempt profitable, and there were no facilities at that time for carrying on some other types of work which seemed necessary to gain an insight into the problem of functional organization of the complex mechanisms of the anterior or cephalic end of the central nervous axis. The problem of functional organization seemed, however, the most intriguing one which presented itself, and a search was made for

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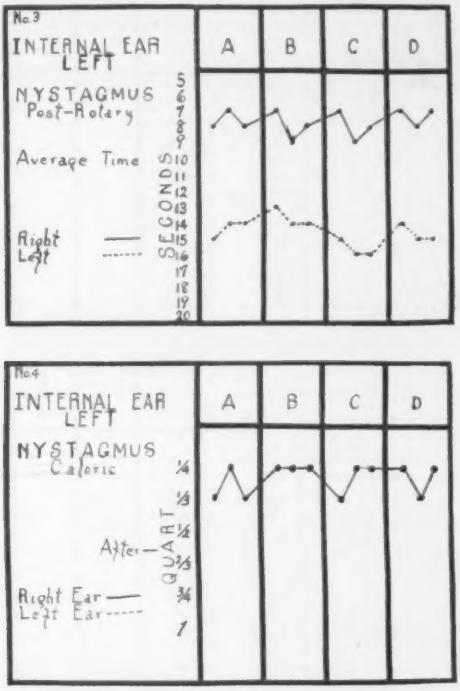
some mechanism which might offer some hope of successful conquest or, at least, sufficiently successful to give some insight into the problem of functional organization in general. Some early attempts to understand the mechanism for respiratory movements led to the selection of the neuromuscular mechanism for the control of the movements of respiration as the one on which the first attack should be made. The results of this attack were encouraging, almost in greater measure than had been hoped for. Comparatively little has been published of these experiments,[2] but the insight gained from them has been of the highest value in the attempt to gain an insight into the functional organization of other mechanisms in the central nervous system.

A brief statement of what we understand by functional organization may be in order. It is now some decades ago that physiologists began to recognize that nervous reactions, or reactions involving the nervous system, were essentially reflex in nature, *i. e.*, some impulse has to come in from the periphery over some afferent path to the central nervous system; it is communicated in the central system to some ganglion cells or origin of efferent fibres, inciting these to activity. The nervous impulses set up in the central ganglion cells are transmitted over these efferent fibres up to the peripheral effectors. Central ganglion cells do not react without due excitation, and this excitation comes in, directly or indirectly, from the periphery. The central cells which are excited to action are those about which the afferent fibres make their terminal arborizations, and the resulting reaction bears a definite relationship to the peripheral receptor which is stimulated and the manner in which it is stimulated. There is a definite nervous mechanism underlying any nervous reaction.

So far as the respiratory movements are concerned, there are certain fibre tracts and central nuclei which are always involved in a natural, spontaneous inspiration and expiration, occurring in an animal without lesions of the central or peripheral system, whose function cannot be taken over by any other fibre tracts or central nuclei. Respiratory movements can continue when some parts of the peripheral mechanism are injured by accident, disease or experiment, the work of the lost parts being taken over by the remaining parts of the normal mechanism for these movements, and not by any other parts of the nervous system. The afferent impulses from the lungs and the respiratory muscles find their way to definite central nuclei, and other impulses going to other central nuclei do not give rise to the same effects. Our problem is to find all the

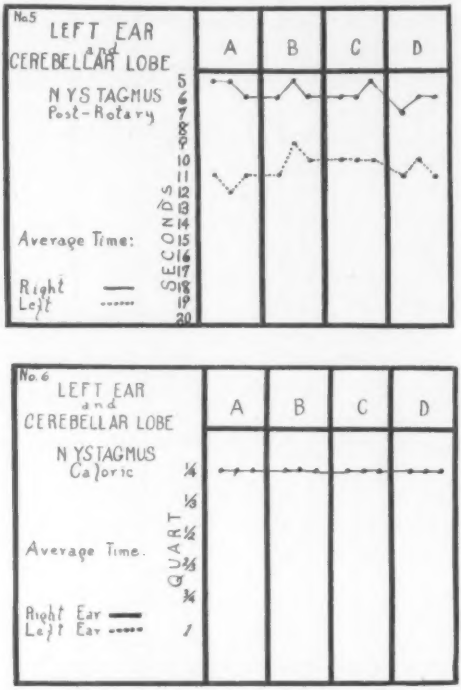
afferent channels which are ordinarily concerned in the initiation and control of respiratory movements, the central nuclei to which they are conducted by the afferent fibres, the manner in which these central nuclei are connected with each other so that the motor discharge is an orderly and adequate response, and the efferent fibres to all the muscles concerned in respiratory movements. The search is not yet ended, but enough has been found to give some idea of



how the mechanism acts, or to show what its functional organization is. But to give a statement of the functional organization of the mechanism for respiration would leave no time for even an unsatisfactory discussion of vestibular nystagmus.

The peripheral receptor from which the impulses leading to the ocular movements which we designate as vestibular nystagmus is, as now seems reasonably clear, the group of nerve endings in the nonauditory portion of the internal ear. A receptor in general is a

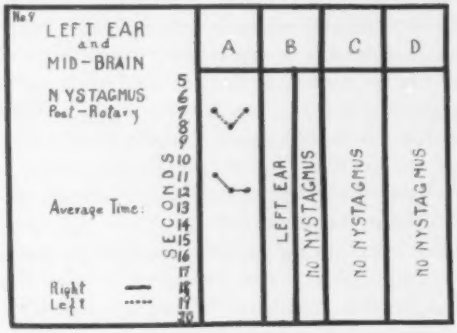
mechanism for transforming some type of physical energy or chemical energy in such a way that it reappears as a nervous impulse. Light falling on the retina is transformed in some way which we do not understand. We cannot follow it as light beyond the retina. The only other manifestation which we have been able to show objectively is the action current—the electrical disturbance which accompanies the passage of an impulse along a nerve, in the optic

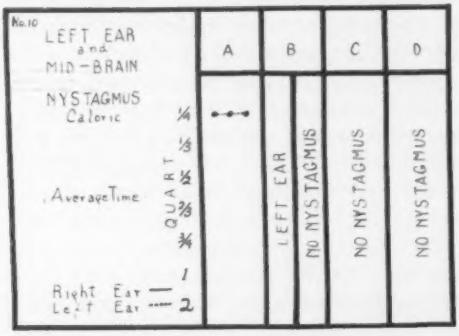


nerve. The production of a small amount of carbon dioxid and a small quantity of heat have been demonstrated in other nerves. Whatever the type of energy which acts upon a receptor, it disappears somewhere in the mechanism of the receptive apparatus, and only the action current can be demonstrated in the nerve fibre beyond it. In general, a receptor of a particular type renders the excitation of its nerve fibre by one and only one type of energy easier than it would otherwise be. Just as light is the type of energy which nor-

mally excites the optic nerve, so the mechanical pressure of fluid is the normal and biologically adequate stimulus for the endings of the vestibular nerve.

There is a widespread opinion or belief that actual fluid currents are necessary for the stimulation of the vestibular endings, but there seems to be no good reason for it. Not only do the currents seem highly improbable, even impossible under ordinary conditions from





the point of view of theoretical physics, but excitation of the endings in the ampulla of the horizontal canal of the shark has been obtained under circumstances which seem definitely to preclude the occurrence of currents in the fluid of the semicircular canals. The vestibular endings seem better adapted anatomically than any others to excitation by pressure, or changes of pressure, of fluids and otoliths within the nonauditory portion of the otic labyrinth. Pressure and changes

of pressure are transformed somewhere in the vestibular afferent endings and reappear as a nervous impulse.

The general results of excitation of the afferent endings of the vestibular nerve are the whole series of changes in position of the eyes, and changes in attitudes of other portions of the body. There seems to be no other mechanism which will give equally reliable information as to attitude (aspect) or change of attitude (aspect) under all conditions of the head in space. But the vestibular endings, and the vestibular system generally, are no more infallible than other systems, and the organism sometimes finds itself in circumstances under which it is difficult to decide between conflicting and diametrically opposed sets of conditions, one real and one apparent. While the inebriated gentleman may merely close one eye in order to decide which of the two possible keyholes is the one into which his key must fit, it is not possible for one merely by closing one ear to resolve the difference between the real and the apparent movement of himself and the surrounding scenery.

The vestibular branch of the eighth cranial nerve, with its cells of origin in Scarpa's ganglion, is the afferent nerve from the vestibular endings to the medulla oblongata. There are five cellular masses within the central nervous system which are the central end stations of the vestibular fibres. These are: *1*. the medial or principal nucleus, *2*. the descending or spinal nucleus, *3*. the superior nucleus of von Bechterew, *4*. the lateral nucleus of Deiters and *5*. the cerebellum. But from here on, the trail becomes much less clear. Nerve fibres are strangely uncommunicative as to their origin or destination when observed in stained sections, and they are even less communicative, if that is possible, as to the nature of the impulses they carry. Anatomists were familiar with the appearance of the dorsal and ventral roots of the spinal nerves long before Bell and Magendie and Panizza showed which ones conducted impulses towards the central nervous system, and which ones away from it. It was long years later before we came to know that proprioceptive impulses are conducted from the spinal cord to the cerebellum by the dorsal and ventral spinocerebellar tracts. And we are still to be informed concerning the peripheral receptors from which arise the impulses passing from the thalamus to the corpus striatum. Anatomical methods alone have certain limitations, and experiment is long and tedious. It may even be complicated.

Some of the anatomical statements leave the experimentalist in a state of bewilderment. Divers anatomists have at sundry times commented upon the relatively small number of fibres in the pyra-

midal tracts, and have wondered how so few fibres could exercise control over such a great mass of muscle as the skeletal musculature includes. Apparently forgetting this perplexity in the next moment they may state, without any reservation whatsoever, that such mere traces of fibres, compared to the pyramidal tracts, as the lateral and ventral vestibulospinal tracts may, can or do influence all the skeletal musculature without any aid or encouragement from other descending pathways. The experimentalist is prone to wonder why, if the relatively great mass of fibres of the pyramidal tract should lead anyone to question its efficacy, the relatively insignificant vestibulospinal tracts should pass without any similar scrutiny of their passport.

The present recrudescence of the work on vestibular nystagmus is to be regarded as a by-product of other work which has involved a closer study than had previously been made of the general mechanism for the initiation and control of the movements of the skeletal muscles. Some years ago work on the convulsive states was started in the laboratory, and it soon became apparent that one part of the problem of the convulsive states was the localization of the cells or origin of the nervous impulses whose motor manifestation was the movements of the skeletal and other muscles during the convulsive seizure. The whole problem of the localization of motor function in the central nervous system and the mechanism of compensation for injury to any one or more portions of the motor system became of importance for the problem of the convulsive states.[8] The experimental work took the form of making operative lesions in various parts of the central nervous system and allowing the animal to recover, sometimes for a year or more. In some of the animals, a second or even a third lesion was made after recovery from the first, and the effects on the animal noted. Some of these experimental lesions involved the internal ear of one or both sides. The results of combined lesions of two or more different structures, peripheral receptors or central nuclei or tracts, were often puzzling, and often full of surprises.

As an illustration, we may cite the case of one cat which had recovered from a cerebellar lesion so completely that, at the end of a year, the only noticeable symptoms were a fine tremor of the head when the animal looked intently at an object some distance away, and a slight impairment of gait, which would escape any ordinary observation. One internal ear was then removed by trephining directly down through the otic bone and taking a core out of the central portion of the otic labyrinth. The animal recovered from this,

and showed only the torsion of the head as a residual sign at the end of three months. The second otic labyrinth was then extirpated. The tremor of the head which had followed immediately upon the cerebellar injury returned, and the animal never recovered anywhere nearly so completely as is usual after bilateral extirpation of the otic labyrinth. We had come upon another instance of the general principle that the results of combined lesions of two or more nervous mechanisms are usually greater than the sum of the residual effects of the lesions considered singly, particularly when the various structures injured are parts of the same functional mechanism. This particular experiment impressed upon us the importance of the vestibular system in the recovery from lesion of the cerebellum. The converse is probably also true, that the cerebellum is of importance in the recovery from lesions of the vestibular system.

Another experiment is of interest in relation to the problem of the central connections of the vestibular nerve and vestibular function. A median incision of the midbrain, passing from the splenium of the corpus callosum backward between the corpora quadrigemina to about the level of the mid-portion of the inferior corpora, was followed by a torsion of the head to the right in the recovery period. In the experiments on median incision of the midbrain, a slight deviation to one side usually gives rise to a torsion of the head to one side, and sometimes to inequality of the pupils if the nucleus of the third nerve is injured on one side. In strictly midline incisions, the head is held in its normal position. Even though such a lesion severs the decussation of the rubrospinal tract, the animal has no difficulty in maintaining itself erect, and never seems to be in difficulty about orientation after the first few days following the operation. The pyramidal tract is not injured during the operation, and the animal regains the ability to walk about. In the particular animal in question, the right internal ear was extirpated some months after the lesion of the midbrain. The torsion of the head was extreme after the recovery from the vestibular operation, so that the line, through the ears, was vertical, with the left ear pointing straight up. If one stood on the right side of the cat and called to it, even though the sound came from the right side, the animal invariably turned to the left in answering the call.

The number of possible combinations of two or more structures, lesions of which might be made either simultaneously or at intervals, is greater than one might suppose. If one starts with the peripheral afferent nerves, passes along the afferent system in order to the cerebral cortex and back again to the periphery, one may easily pick out

100 different central nuclei or fibre tracts, without going into any great anatomical detail. If these hundred structures are arranged in order in a vertical column at the left of a chart, they may be listed in the same order at the top of the chart. For every structure in the vertical column at the left, one has a choice of any one of the 99 other structures for the second lesion. It is merely a problem in grammar school arithmetic to show that there are more than 10,000 possible combinations of this sort possible in the nervous system, and more than a hundred in which the vestibular system becomes one of the pair. I trust that we may be pardoned for not having worked out all of the possible combinations, for if one goes through the literature, there is an astonishing lack of information. At present, the student of the functional organization of the nervous system has an almost unworked field of great extent before him.

The routine procedure in the experiments has been to make some one operative lesion under aseptic precautions and allow the animal to recover, some weeks or months later, one femoral vein is exposed under local anesthesia and absinthe injected intravenously, beginning with low doses and gradually increasing until a convulsion is elicited. The dose of the convulsant drug is further increased until the lethal dose is reached, after which a general postmortem examination of the animal is made, and the brain and spinal cord removed for histological examination. Peculiar ocular reactions are sometimes observed and, in the interval immediately following the clonic manifestations of a convulsion, typical nystagmus is sometimes observed. It may be vertical, horizontal or rotary. When lateral (horizontal) nystagmus occurs, there is usually an inequality of the pupils and the quick movement is generally to the side of the larger pupil. There have been a few exceptions in which the quick component was directed toward the smaller pupils and we have seen one experiment in which the direction of the quick component was at first toward the side of the larger pupil and later on toward the side of the smaller. This inequality of the pupils and nystagmus may occur in control animals which have had no lesion, or at least no experimental lesion, of the nervous system. We do not know the significance of these ocular movements, but we are now fairly well convinced that the convulsant drug, absinthe or camphor monobromid, acts upon the cerebral cortex of the experimental animal.

Some time between the experimental operation and the injection of the absinthe, various tests are made upon the animal, one of which has been the examination of the vestibular reactions. Some peculiar and unexplained results have been found. For example, in

one animal, with a definite puncture wound of the head of the caudate nucleus, rotation was not attended by any unusual result. Running cold water into the ear of the same side as the lesion set up a convulsive tic of the head, face, eyelids, for all the world like some we have seen which have been diagnosed most emphatically as tics of psychogenic origin. The observations on this animal could not be repeated as it died about forty-eight hours afterward, shortly after the subsidence of the tic. Postmortem examination did not reveal any gross condition which would explain the animal's death.

We wish here to give only the results of rotation and the introduction of cold water into the ears at varying intervals after experimental operations in a group of animals. Pending the results of histological examination of the brains of such animals as have died, the location and extent of the lesion is stated in terms of the gross appearances. Some animals are still living. In such cases, the location of the lesion is stated in terms of experimental probability.

Vestibular nystamus is an easily observed phenomenon following rotation and caloric testing of mammals. The relation of the planes of the induced nystagmus to the planes of the semicircular canals and the direction of the quick and slow components brought about by the method of stimulation are constant factors that permit easy observation of variations from the normal.

It is significant that, in a normal animal, the nystagmus time will be the same, within narrow limits, after right and left rotation, though in a given animal that time may be near the upper or lower limit for that species.

The quantitative values in nystagmus, *i. e.*, the exact degree of stimulation necessary to induce it, its amplitude, the speed of the quick and slow components, are not so easily detected as are the qualitative variations from the normal.

There is no simple method by which one may measure the amplitude and speed of nystagmus. Experience, however, in testing animals enables one to estimate rather accurately these quantitative variations in terms such as below average, average or above average.

Procedure in All Experiments: 1. Postrotary nystagmus was observed as to its time, direction, plane, amplitude and speed after rotation of twenty times in twenty seconds. The head was held in its anatomical position and the eyes were not shaded while being rotated. 2. In the caloric test, the time of the appearance of the nystagmus was taken following irrigation of the ear, along with the direction, plane, amplitude and speed of the eye movements. The

temperature of the water used was 42-45° F., and the flow was at the rate of one quart in two minutes.

Experiment 1. Normal or Control Cats. Charts 1 and 2: Three animals were used for rotation and caloric testing at weekly intervals for four weeks. The average postrotary nystagmus time on right turning was 14.4 seconds; on left turning, 14 seconds. Eleven seconds was the lower limit observed in one animal and eighteen the upper limit observed in another animal. Those results are approximately the same as those observed by Prince[1] on postrotary nystagmus time in cats. He also learned that the time remained fixed for each animal within narrow limits, after testing a number of animals fifty to sixty times daily for twenty consecutive days. We observed, in the caloric tests, that an active nystagmus was produced in the normal animals following irrigation with one-half to two-thirds of a quart of water or in 30 to 45 seconds.

Experiment 2. Lesion. Left Internal Ear. Charts 3 and 4: There appeared a nystagmus, slow component to the left and quick component to the right, on recovering from the anesthetic. The nystagmus persisted for four days. This is in agreement with previous observations.[5, 6]

Postrotary Nystagmus: Time: right, eight; left thirteen; type: right, normal; left normal; amplitude: right, average; left, average; speed: right, average; left, above average.

Caloric Nystagmus, Right: Quantity*: one-fourth to one-third; type, normal; amplitude, average; speed, above average.

Postmortem: Left internal ear destroyed.

Experiment 3. Lesion. Bilateral Inner Ears (at the same time): There were a few conjugate eye movements on recovering from the anesthetic. Those ocular movements were ataxic in character; being definitely different from the nystagmus after unilateral ear destruction. There was no nystagmus after right or left rotation. Wilson and Pike.[5]

Experiment 4. Left Internal Ear and Left Cerebellar Lobe. Charts 5 and 6: There appeared a vestibular nystagmus, slow component to the left, quick component to the right on recovering from the anesthetic.[2] One got the impression that it was a little greater in speed than usual following vestibular lesion and above. It persisted about six days.

Postrotary Nystagmus: Time: right, six; left, eleven; type: right, normal; left, normal; amplitude: right, below average; left, below average; speed: right, above average; left, above average.

*Quantity is in part of a quart used.

Caloric Nystagmus, Right: Quantity: one-fourth; type: normal; amplitude: above average; speed: above average.

Postmortem: Gross appearance; practically all the left cerebellar lobe was removed, but it is questionable whether the dentate nucleus was injured.

Experiment 5. Lesion: Motor Areas and Left Internal Ear. Charts 7 and 8: The ear ablation was two months after the operation on the motor areas. Spontaneous vestibular nystagmus appeared after the ear ablation and did not seem to have been modified by the preceding cerebral operation.

Postrotary Nystagmus: Time: right, eight; left, fourteen; type: right, normal; left, normal; amplitude: right, average; left, average; speed: right, average; left, average.

Caloric Nystagmus, Right: Quantity: one-fourth to one-third; type: normal; amplitude: average; speed: average.

At times the amplitude was above average and speed was below average.

Experiment 6. Lesion: Midbrain. Left Internal Ear. Charts 9 and 10: The ear operation was done one month after the midbrain lesion. There was no spontaneous nystagmus following the midbrain incision. The occiput rotated to the right to a greater degree than the rotation observed after right ear ablation. On recovery from the anesthetic given for the left ear operation there appeared a horizontal nystagmus, large amplitude and slow speed. It was impossible at times to distinguish a quick and slow component.

The usual rotation of the occiput to the left occurred after the left ear operation, apparently not influenced by the midbrain incision. Findings on testing following midbrain lesion:

Postrotary Nystagmus: Time: right, twelve; left, seven; type: right, normal; left, normal; amplitude: right, average; left, average; speed: right, average; left, average.

Caloric Nystagmus, Left: Quantity: one-fourth; type, normal; amplitude: average; speed: average.

There was no nystagmus induced on rotation or caloric test after the left internal ear operation.

Postmortem: Incision extended from splenum to pons and on the left side of the aqueduct.

Experiment 7. Lesion. Left Cerebellar Lobe and Vermis. Charts 11 and 12: There was no spontaneous nystagmus or rotation of the head after the operation as was noted on animals having unilateral inner ear lesion. Few conjugate ocular movements?

Postrotary Nystagmus: Time: right, twenty; left, twenty-two; type: right, normal; left, normal; amplitude: right, below average; left, below average; speed: right, above average; left, above average.

Caloric Nystagmus: Quantity: right, one-fourth; left, one-fourth; type: right, normal; left, normal; amplitude: right, below average; left, below average; speed: right, above average; left, above average.

The increased excitability of the vestibular nerve, which appeared to be slightly greater on the left side, lasted three months, the longest time of postoperative observation.[4]

Experiment 8. Lesion. Left Temporal Lobe. Charts 13 and 14: No spontaneous nystagmus occurred following the operation.

Postrotary Nystagmus: Time: right, seventeen; left, fifteen; type: right, normal; left, normal; amplitude: right, above average; left, above average; speed: right, average; left, average.

Caloric Nystagmus: Quantity: right, two-thirds; left, one-third to one-half; type: right, normal; left, normal; amplitude: right, average; left, average; speed: right, average; left, above average.

Postmortem: Gross appearance: Incision extended the length of the temporal lobe with destruction of the lobe excepting posterior inferior portion.

Experiment 9. Lesion. Left Temporal Lobe. Left Motor Area. Charts 15 and 16: The left motor area operation was done one month after the left temporal lobe operation. There was no spontaneous nystagmus following either operation. Findings on testing following left temporal lobe lesion:

Postrotary Nystagmus: Time: right, eighteen; left, seventeen; type: right, normal; left, normal; amplitude: right, above average; left, above average; speed: right, average; left, average.

Caloric Nystagmus: Quantity: right, two-thirds; left, one-fourth; type: right, normal; left, normal; amplitude: right, average; left, above average; speed: right, average; left, above average.

Findings on testing following left motor area operation on the same animal:

Postrotary Nystagmus: Time: right, twenty; left, seventeen; type: right, normal; left, normal; amplitude: right, above average; left, above average; speed: right, average; left, average.

Caloric Nystagmus: Quantity: right, two-thirds to three-fourths; left, one-fourth to one-third; type: right, normal; left, normal; amplitude: right, average; left, above average; speed: right, average; left, above average.

SUMMARY.

1. Vestibular nystagmus occurred postoperatively in the animals of this series of experiments only when the vestibular nerve was involved in the lesion. There were a few to and fro conjugate eye movements following some of the cerebellar lesions, but those could not be mistaken for a vestibular nystagmus.

2. Destruction of one inner ear was followed by: *a.* Postrotary nystagmus time of approximately half normal duration after turning in direction of the intact ear. *b.* Slower eye movements after turning in direction of the intact ear than in opposite direction. *c.* Earlier and more active caloric response in the intact ear than normal. Those findings were observed not only just following operation on the inner ear but also in two animals tested one year following unilateral destruction of the inner ear. That was the longest time after operation that any observations were made.

3. Destruction of one inner ear and cerebellar lobe, same side. The addition of the cerebellar injury to the ear lesion changed the nystagmus elicited from the ear lesion alone, only in that the amplitude was below average, speed above average, and there was less difference in the speed of the two components.

4. Destruction of one inner ear and both motor areas. No additional change over the ear lesion alone was noted in the nystagmus following the motor area lesions.

5. Midbrain lesion slightly to the left of the Sylvian aqueduct and extending from the splenium of the corpus collosum to the pons. The nystagmus response was comparable to that after right ear destruction.

6. Destruction of cerebellar lobe and about half of vermis. Postrotary nystagmus time was definitely longer than normal, amplitude less than average, speed above average with no essential difference in the speed of the components. The ear on the side of the lesion showed an earlier response than normal for caloric testing.

7-8. Unilateral cerebral lesions: temporal lobe destruction not involving the thalamus and motor area destruction without injury to the striate. There was a temporary increase in the nystagmus response to rotation and caloric test, and slightly greater on the side of the lesion. The findings on testing after the addition of the motor area lesion to the temporad lobe lesion were nearly the same as those observed following only the temporal lobe lesion.

It is interesting to note in this investigation that we have not observed the slow ocular movements of vestibular origin without the

quick component being present, or any dissociated eye movements, or any abnormal types of nystagmus with reference to the planes of the semicircular canals.

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Columbia University.

OTOLARYNGOLOGIC EXAMINATION OF 935 "NORMAL" INDIVIDUALS.*

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"Der Normalbegriff ist ja ein wahrer Proteus und es ist nicht leicht, in den vielen Gestalten, die er annehmen kann, den Kern seines Wesens zu erkennen," p. 112[1].

That the number of contributions to the medical literature dealing with the *normal* individual is regrettably small is well known. And the importance of the existence of such contributions is obvious. It is the object of this paper not only to add another set of findings to the bibliography of normals but to encourage the publication of more.

The basic material presented here is a part of the enrollment records of 935 healthy individuals that agreed to participate in the John J. Abel research on the common cold now being conducted at the medical center of the Johns Hopkins University. The individuals belong to either of two groups. The *medical group* contains about 350 persons. Approximately two-thirds of these are medical students, and about one-third are students or staff members of the School of Hygiene and Public Health. The remaining individuals are members of the second group, namely, the *family group*, which consists of about 100 families living in Baltimore.

The age distribution of both sexes of the two groups combined is shown in Table I. The age range has a lower limit of a few months, and an upper limit of 81 years. The age group 20-29 years with a total of 368 has the largest number of representatives. The groups 0-4 and 30-39 are next in order of size, each with 148 individuals.

In Table II are recorded the various findings together with the number and per cent of the individuals associated with each. In addition, the number of persons for whom no data were available is also recorded under each anatomical part included in the examination. The 935 individuals were divided into two age groups, one under fifteen, and the other fifteen years and over. There are 275 individuals in the first group and 660 in the second. The younger age

*From the John J. Abel Fund for Research on the Common Cold, and the Department of Biostatistics (Paper No. 162), School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, Maryland.

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The sinus roentgenograms were read by Dr. F. H. Baetjer, professor of Clinical Roentgenology. The remaining examinations were performed by Dr. J. J. Chisolm and Dr. S. W. Egerton, associate and instructor in Clinical Laryngology, respectively.

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TABLE I.

Age-sex distribution of the individuals examined.

Years	*Males*	*Females*	*Both Sexes*
Total	*552*	*383*	*935*
0- 4	87	61	148
5- 9	37	35	72
10-14	22	32	54
15-19	17	17	34
20-29	263	105	368
30-39	74	74	148
40-49	36	33	69
50-59	8	14	22
60-69	4	6	10
70-79	3	5	8
80-89	0	1	1
Unknown	1	0	1

TABLE II.

Otolaryngologic findings in 935 "normal" individuals. The finding that gives rise to per cents in the two age groups that are significantly different is followed by an asterisk.

Part Examined	*Finding*	*Individuals, Less Than 15 Years of Age, Both Sexes*		*Individuals, 15 Years of Age and Over, Both Sexes*	
		1 Number	*2 Per Cent*	*3 Number*	*4 Per Cent*
Sinuses (by X-ray)	Clear*	105	68.8	437	83.2
	Cloudy*	48c	31.2	88e	16.8
	Not taken	121		135	
Glands at angle of jaw	Small	121	70.3	369	68.5
	Large, or moderate	51	29.7	170	31.5
	Not noted	103		121	
Teeth and gums	Good, i.e., no infection	148	83.6	420	77.9
	Poor	29	16.4	119	22.1
	Not stated	98		121	
Tonsils	Small, normal*	29	16.8	133	24.6
	Small, inflamed	7	4.0	27	5.0
	Moderate, normal*	23	13.3	26	4.8
	Moderate, inflamed*	22	12.7	29	5.4
	Enlarged, normal	11	6.4	18	3.3
	Enlarged, inflamed*	29	16.8	37	6.8
	Partially removed, normal	5	2.9	60	11.1
	Partially removed, inflamed	2	1.2	8	1.5
	Removed at 12 or before	43	24.9	54	10.0
	Removed after 12 years of age	1	.6	137	25.3
	Removed, age unknown	1	.6	12	2.2
	No examination	102		119	
Pharynx	Inflamed or injected*	13	7.6	186	34.9
	Normal*	157	92.4	347	65.1
	Unknown	105		127	

Ear drums	Scarred (one or both)	15	8.5	56	10.6
	Normal[a]	161	91.5	472	89.4
	Unknown	99		132	
Nasal septum	Deviation, spur or ridge*	34	21.9	338	71.9
	Normal*	121	78.1	132	28.1
	Unknown	120		190	
Breathing space	Both sides poor*	3	1.7	26	4.8
	One side poor*	2	1.1	78	14.6
	Both sides good or fair*	169	97.1	432	80.6
	Unknown	101		124	
Sinuses (by transil.)	Clear*	138	95.2	482	89.2
	Cloudy*	7d	4.8	58f	10.7
	Not done	130		120	
Turbinates	Abnormal*b	5	4.5	121	23.7
	Normal*	107	95.5	389	76.3
	Not stated	163		150	
Adenoid tissue	Small, normal*	27	21.6	204	39.2
	Small, inflamed	8	6.4	48	9.2
	Small, unknown	2	1.6	9	1.7
	Moderate, normal	12	9.6	24	4.6
	Moderate, inflamed	11	8.8	20	3.8
	Moderate, unknown	4	3.2	4	.8
	Large, normal	10	8.0	20	3.8
	Large, inflamed	7	5.6	20	3.8
	Large, unknown	4	3.2	2	.4
	Removed or non-visible	40	32.0	169	32.5
	Not noted	150		140	

a. Including opaque or retracted drums.
b. Polypoid, atrophic, or edematous.
c. Antra: 45; frontals: 0; antra, frontals and ethmoids: 3.
d. Antra: 5; frontals: 0; antra and frontals: 2.
e. Antra: 87; frontals: 0; antra, frontals and ethmoids: 1.
f. Antra: 51; frontals: 2; antra and frontals: 5.

group data are listed in columns one and two while columns three and four contain the data of the older group. The different terms describing the findings are self-explanatory. For example, "sinus X-ray clear" indicates that the antra, frontals, and ethmoids as shown by the plate were found clear by the roentgenologist, while "cloudy" includes all plates that were doubtful and those that showed at least one cell cloudy; "sinus transillumination clear" indicates that the antra and frontals of the individual were found clear by the transilluminator, and "cloudy" was used when one or more cells gave evidence of cloudiness; and so on.

Facts of interest that a careful reading of Table II will not disclose may be brought to light by comparing statistically the per cents yielded by one age group with those of the other. Such a comparison reveals one or more findings under each of the following anatomical parts that are significantly different: sinuses (by X-ray), tonsils, pharynx, nasal septum, breathing space, sinuses (by transillumination), turbinates, and adenoid tissue. Findings showing significant

differences have been marked with an asterisk in the table. The differences presented by the tonsils and the adenoid tissue, respectively, agree with what is known concerning the association of lymphoid atrophy with the aging process. The pharynx, nasal septum, breathing space, and turbinates, on the other hand, occur abnormally more frequently in the older age group. This is not surprising since a deviation from normal is not only more difficult to detect in infants and children but obviously requires some time to develop. Some difficulty is encountered when it is attempted to explain the per cents yielded by the sinuses under X-ray and transillumination. The X-ray shows a larger per cent of the older group *clear* while the reverse is given by transillumination. The difference by transillumination is, perhaps, best explained in general by anatomical differences. The otolaryngologist frequently experiences the transillumination difference just recorded and has thus been forced to place less confidence in the transillumination of children than of adults. An attempt to explain the difference by X-ray would, with only the material thus far presented available, result in mere guessing[2].

The question of whether or not there is any association between the various findings now arises. It was decided to deal with each age group separately and accordingly two tables[3] were constructed. The two columns of Table II, *Part Examined* and *Finding*, became the marginal column of each of the two tables, and the same material also formed their horizontal margin. Thus the first column of both tables contains the findings of all of those individuals having a clear sinus X-ray, the second column of those with a cloudy X-ray, and so on. It is now possible to determine for each age group the effect on the different findings of holding a particular finding constant. That both tables are made up of a number of smaller tables that are of the four-fold type is clear. For example, the following four-fold table (Table III) was removed from the large table of the older age group:

TABLE III.

Glands and turbinates. Individuals of age 15 years and over.

Turbinates	*Glands at Angle of Jaw*	
	Small	*Large*
Abnormal	21% (73)*	30% (47)
Normal	79% (280)	70% (108)

*A number in a parenthesis indicates *number* of individuals.

An analysis of this table indicates that individuals of age 15 years and over with small glands at the angle of the jaw are more likely to have normal turbinates than those whose glands are large.

The significant associations[4] which an analysis of the different four-fold tables revealed may be given under three headings, thus:

A. Individuals of All Ages: 1. With small normal tonsils are likely to have small glands at the angle of the jaw and a small amount of normal adenoid tissue.

B. Individuals Under Fifteen Years of Age: 1. With good teeth and gums are likely to have clear sinus X-rays and small glands at the angle of the jaw. *2.* With good or fair breathing space are likely to have clear sinus X-rays. *3.* With a small amount of normal adenoid tissue are likely to have small glands at the angle of the jaw and an abnormal nasal septum.

C. Individuals Fifteen Years of Age and Over: 1. With small normal tonsils are likely to have a normal pharynx and normal turbinates. *2.* With an abnormal nasal septum are likely to have small glands at the angle of the jaw. *3.* With good or fair breathing space are likely to have a normal pharynx and a normal nasal septum. *4.* With normal turbinates are likely to have small glands at the angle of the jaw and a normal pharynx. *5.* With a small amount of normal adenoid tissue are likely to have clear sinus X-rays and a normal pharynx.

SUMMARY.

1. The otolaryngologic findings in 935 "normal" individuals of both sexes divided into two age groups are recorded.

2. The findings of the two age groups are compared.

3. The holding of various findings constant leads to the determination of the probable presence or absence of association between different findings.

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2. The whole subject of the reliability of the transillumination and the X-ray of the sinuses is fraught with difficulties and deserves a detailed analysis. Such an analysis is, in fact, in preparation and will be published separately.

3. The unusual size of these tables prohibits their inclusion here.

4. A four-fold table that yielded a *chi-square* of five or *more*, or in other words a probability of .025 or *less*, was considered as disclosing a significant association.

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UNRECOGNIZED AND LATENT MASTOIDITIS WITH LATERAL SINUS THROMBOSIS.

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Otitis media, presenting such severe gastrointestinal, pulmonary and other general symptoms as to cause the physician to overlook the real seat of trouble, has been known for a long time, particularly in infants and young children. Hartmann[1], in 1898, demonstrated that free drainage from the middle ear relieved these severe disturbances manifesting themselves elsewhere in the body. Alden and Lyman[2], Byfield[3], Floyd[4], McDougall and Knauer[5] and others claim that athrepsia, infantile diarrhea and other severe and sudden gastrointestinal disturbances may be directly caused by a middle ear infection. Marriott[6] and Dean[7] found only infected mastoids at autopsy of some infants dying with the symptoms just mentioned; and Rendu[8] found at autopsy bilateral purulent mastoiditis in 90 per cent of a series of 19 infants not presenting the classical symptoms of mastoiditis during life. It was Le Mee[9] who called these cases "unrecognized" in contradistinction to latent and primary mastoiditis; for he claimed that on thorough examination the drumheads are found full, even bulging, and without lustre; and paracentesis reveals pus. Older children may complain of a little pain, fullness or impaired hearing.

Asherson[10] observed that the sagging or bulging of the posterior superior wall of the osseous meatus may be the only symptom present. Another important sign is the rapid accumulation of pus in the meatus constantly or periodicallly.

That mastoiditis may also proceed unrecognized in adults on account of the prominence of other symptoms, is well illustrated in the second of two striking examples of this condition cited below.

Case 1: R.A., a boy, age 11 years, gave the following history: Had frequent colds until he was 7 years old, when a T. and A. operation was done. The following year he developed a streptococcus hemolyticus mastoiditis on the right side, which was operated on with uneventful recovery. March 31, 1925, three years after the mastoidectomy, the child began to have slight pain, and some swelling over the mastoid scar. The drum membrane was dull, opaque and somewhat bulging. A paracentesis was done and mucopurulent matter obtained. The swelling over the scar did not subside but gradually increased

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and, on April 8, it was opened and the antrum drained. On April 14, the other ear became infected. The drum membrane was red and bulging. A myringotomy was done and free pus obtained. For six days the patient ran the usual course of double otitis media. The temperature never rose over 100°F. and there was little or no pain, but a fair amount of discharge from the left ear. At the end of this time the patient suddenly took a turn for the worse; temperature rose to 102°F., pulse 110, and the discharge from the left ear became scant. The patient was immediately taken to the hospital where he was examined with the following findings: White blood count, 21,000, with 88 per cent polymorphonuclears; blood urea, 46.73; blood culture, negative; blood pressure, 150/90; urine: Specific gravity, 1.018; smoky color, cloudy appearance, acid reaction; albumin, ++; occult blood, +; many coarse and fine granular casts; sugar, negative. The internist consulted, decided not to have the mastoid process opened since there was absolutely no tenderness or swelling and X-ray examination showed no cell destruction. The patient ran a stormy course for the next nine days. The temperature rose at times to 105.4°F. The urine increased in cloudiness and at times it looked like diluted blood. There was some edema around the eyes and ankles, severe headache and querulousness. The left ear, which presented a large sized opening in the lower outer quadrant of the drum membrane, was discharging very little. Suction and irrigations were used constantly without effect until the tenth day, when the patient woke up with an entirely changed appearance. Temperature was normal, the urine became almost clear—no blood, but a slight amount of albumin was present. At the same time it was noticed that the left ear canal was filled with a thick creamy pus. Upon cleaning this out, the drum was found to be unchanged.

The temperature rose, however, two days later to 102°F. Again the urine showed blood and casts. The opening in the drum was extended upwards to and including Schrapnell's membrane, and suction was used at the same time. The temperature dropped to normal again, and the urine became almost clear. From then on the course was as follows: after a profuse discharge for a day, the urine was clear, the temperature normal, and the appearance of the patient good. This lasted for three or four days, when the urine began to show a slight trace of albumin becoming more pronounced, and the temperature slowly rose intermittently to about 100.5°F. when another gush of pus appeared and the patient's condition returned to normal. These cycles continued periodically every eight or nine days for the next two months. During this time the patient was kept in bed under

rigid treatment for his nephritis, with regular irrigations of both ears. The right mastoid opening, which still had some scant discharge, was dressed daily until it was dry, when the wound closed.

Another X-ray of the left mastoid was taken and showed the same picture of a large antrum and no destruction of cells. The opening of the mastoid was considered but deferred on account of the intermittent improvement of the patient.

A left antrotomy was done, however, on June 12, 1925. On incising the skin, the periosteum was found to be normal, and the cortex rather thick. The antrum was of large size, and filled with thick mucopurulent secretion. There were a few small cells surrounding it, with no destruction of bone. The mastoid process was leveled off, the wound sutured and a drain inserted in the lower angle. There was a secondary hemorrhage on the eighth day, after which the patient made a rapid recovery except that the wound closed very quickly, and the urine, which had become free of albumin and casts since the operation, again became slightly cloudy. A rubber tube drain was introduced into the antrum and irrigations through this tube carried out for about four weeks, until the mastoid became completely dry and the patient discharged as cured Aug. 1, 1925. It is now five years since his discharge and there has been no recurrence of the nephritis.

Case 2: B. G., age 40 years, was brought to Sinai Hospital, Feb. 4, 1930, unconscious, with a history of having fallen in this condition while walking on the street. Upon regaining consciousness he was found to have a complete right hemiplegia. This cleared up very quickly and the patient was discharged greatly improved after twenty days' stay in the hospital, with a diagnosis of cerebral hemorrhage, hypertension and right hemiplegia. One month later he returned to the hospital with a right sided weakness. He remained in the hospital for two weeks, condition unimproved and discharged with a diagnosis of brain tumor. A month later he returned to the hospital complaining of headaches. His hemiparesis had entirely cleared up, but he complained of severe headaches and tinnitus in the left ear. His hearing in that ear was also markedly reduced. His mental state was very depressed and he talked of suicide. He was therefore transferred to a psychopathic hospital where a complete study was made with negative findings. He stayed in the last hospital only 10 days and was discharged in a somewhat improved condition. From then on, however, his ear symptoms became very evident. A week after leaving the latter hospital he suddenly noticed a discharge from the left ear with pain and tinnitus, but no tenderness

over the mastoid. Since the aural discharge his mental condition improved greatly, but paracentesis had to be done twice in the following month. The discharge was quite profuse at times. His headache continued and on July 3 he was admitted to the Sinai Hospital with tenderness and swelling over the mastoid. X-ray showed destruction of the cells on the left side. A mastoidectomy was performed on July 16, 1930. We found complete destruction of cells with a great deal of pus in the mastoid and a perisinus abscess. After completing the operation the mastoid cavity was drained. The patient remained in the hospital ten days, making a complete recovery, both as to his ear and mental condition.

These two cases present a type of mastoiditis which is easily overlooked because the severity of symptoms in other regions distracts our attention from the mastoid condition.

However, a true latent mastoiditis has been demonstrated by Gorke and Doderlien[11]. These authors have shown by microscopic specimens at autopsy the presence of acute otitis media with mastoidits in patients who had no symptoms whatever of this condition during life. Krantz[12] also showed lately that latent mastoiditis occurs much more frequently than was formerly thought. Many of the cases of so-called primary mastoidits are probably really latent in character.

Asherson[10] claims that suppuration in the mastoid with intracranial extension may be present without symptoms, and stresses the point that when a patient with a running ear has any symptoms suggestive of dural involvement this history must be regarded seriously in spite of the absence of the symptoms.

The first of the two following cases illustrates not only a definite latent mastoiditis but a latent lateral sinus thrombosis and abscess as well.

Case 3: A man, age 35 years, with a history of a running ear all his life, was otherwise in perfect health. Examination revealed cholesteatomatous masses in the running ear. He was apparently feeling well even with a temperature of 105°F. and the fact that three hours before he had had a chill which brought him to my office. The patient was immediately sent to the hospital and a radical mastoidectomy was performed. A large cholesteatoma was removed from the antrum; the wound was sutured and the cavity packed. The patient made an uneventful recovery. He remained in the hospital for 10 days. The last five days of which he was up and about, recuperating progressively with absolutely no rise in temperature. During the night of the eleventh day the nurse on duty heard a peculiar noise from the direction of this patient. He was found frothing bloody

secretion at the mouth and by the time she called the house physician, he was dead. Autopsy, a few hours later, revealed a well organized lateral sinus thrombosis surrounded by an abscess extending backward two inches into the horizontal limb of the lateral sinus wall and downward to the jugular bulb.

Case 4: E. E., a boy, age 11 years, well developed, came under my observation May 10, 1930, with following findings:

Family history as well as past history negative. A year ago he had an acute otitis media suppurativa in right ear which lasted six weeks; there was no apparent mastoid involvement. He had no further trouble until two days before when he began to have pain in his right ear with fever. Examination revealed some tenderness over the right mastoid, no swelling, canal normal, drum membrane bulging and red. Temperature 104°F. A paracentesis was done, but no pus obtained. Irrigations were instituted but with no effect. The next day his temperature was 105°F. and he was sent to the hospital. The leucocyte count was 7500. The drum membrane was unchanged. There was moderate tenderness over the mastoid. The opening in the drum membrane was enlarged, but still no pus was obtained. The next day the temperature slowly came down to 102.4°F. The leucocyte count was 8300; polymorphonuclears 70 per cent; lymphocytes 22 per cent; large mononuclears and transitionals 6.8 per cent. No eosinophiles or basophiles. The tenderness over the mastoid was less marked. On the third day, March 13, the temperature rose again to 105°F. Leucocytes were 9000 in number, with practically the same differential. A mastoidectomy was performed. The periosteum and cortex were found to be normal. Upon opening the cortex, however, it was found to be thin and a dark colored fluid pus of very foul odor presented itself under pressure. The cells were well developed and the bony partitions quite black. The entire mastoid was cleaned out without exposing the dura anywhere. A culture was made from the pus and also from the blood. The wound partly closed, and was dressed as usual. A gram negative hemolytic streptococcus was grown out of both the wound and blood in about 12 hours. The following day the patient had a chill and his temperature rose again to 105°F. The internal jugular was ligated and the lateral sinus opened in the usual way. The same character of pus with the same odor was found in the lateral sinus with organized clot above extending one inch into horizontal limb, free bleeding was obtained upon removing same. No bleeding obtained from the lower end. The patient was in partial shock and recuperated slowly. The lower end of the sinus drained pus for about four weeks during which time the patient was kept in

bed with the head lowered until the wound completely healed, when the patient was discharged in good condition.

The last case not only proves the possibility of a latent mastoiditis but also the possibility of the existence of a lateral sinus thrombosis for some time without manifesting any symptoms. Tobey[13] claims that there is no typical train of symptoms in early cases of sinus thrombosis. Three groups are mentioned by him: a septic expulsive type (a very acute infection); a typhoidal type (low grade); a latent type having intermittent mild symptoms of septic absorption for weeks and months following a mild acute suppurative otitis media and suddenly developing a virulent meningitis or other symptoms. The last two cases mentioned belong to the third classification.

Friedenwald[14], in a paper entitled "On the Efforts of Nature to Cure Septic Thrombosis of the Lateral Sinus" reviews the literature of afebrile sinus thrombosis and states that a number of cases have been reported in which complete obliteration of the sinus by collapse, or more probably by organized thrombus has been observed, either during the operation or after death. He adds two cases of his own where abscesses existing for a long period of time were found walled off in the lateral sinus with very few symptoms. A number of these cases presented fistulous opening in the sinus walls. The nonfebrile course is explained by him either by the aseptic character of the thrombus, by the mild nature of the infection or by the discharge of pus when formed through an opening in the external wall.

Latent mastoiditis can easily be explained in the course of such severe constitutional diseases as tuberculosis, nephritis and cancer. To this class belong the latent ear infections accompanying the exanthema in children. These latent ear infections during the course of a severe constitutional disease should not be confused with the unrecognized mastoiditis described above, where the constitutional symptoms are the direct effect of the ear infection. Seifert[15] reported a latent mastoiditis with lateral sinus thrombosis followed by death in a patient with chronic alcoholism. There are other cases, however, the latency of which cannot be explained except by Reverchon's[16] suggestion of a special disposition on the part of the patient.

It is sometimes impossible to make a diagnosis in latent mastoiditis; but the condition should be kept in mind in any severe and unexplained illness, particularly when there is a history of some ear trouble even if it was of a very mild character.

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ACUTE SURGICAL MASTOIDITIS FOLLOWING FRACTURE.

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Acute mastoiditis, developing after a skull fracture is a relatively uncommon condition. Vance, in a review of 512 necropsies upon cases of skull fracture, found that 48 died of acute suppurative leptomeningitis or some type of generalized septic infection and that in 18 of these 48 cases the source of infection was through the middle ear and mastoid.

Shemley states that fractures of the base of the skull are often concomitant with fractures of the vault and that about 80 per cent of fractures of the base originate as a linear fracture of the vault and extend to the base. The majority of fractures of the base involve the middle fossa because this is the weakest portion. Statistics indicate that from 40 per cent to 80 per cent of fractures occur here. Politzer, in discussing fractures of the bony meatus says, "as a rule fractures of the meatus are seldom confined to this section, but are usually complicated by fissures of the superior and inner walls of the tympanic cavity, of the mastoid process, of the petrous portion of the temporal bone and of the base of the skull." Fractures of the base most often involve the petrous portion of the temporal bone.

Grove divided fractures of the temporal bone into three varieties, longitudinal fractures, transverse fractures, and avulsion of the tip of the petrous pyramid. The transverse fracture of the pyramid is much less common than the longitudinal type. It crosses the pyramid at its weakest portion and may or may not involve the cochlea. The vestibule is fractured, however, and the membranous portion of the inner ear destroyed. This type of fracture may or may not involve the middle ear. The type of fracture producing avulsion of the tip of the petrous bone is rare and may or may not cause injury to the seventh and eighth nerves at the internal auditory meatus. The most common type fracture is the longitudinal one. Stenger, Ulrich, and Schlonbauer and Brunner state that the longitudinal type of fracture usually runs along the anterior border of the pyramid originating in the middle fossa or region of the Gasserian ganglion and ending in the tegmen tympani or antri and in the external canal. The bony labyrinth often is not involved in this type of fracture and the facial nerve may be involved in the region of its genu or the fracture line may pass anterior to it. In these cases labyrinthine symptoms are

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often absent and there is only slight impairment of hearing. Schonbauer and Brunner presented 23 fractures of the petrous bone, 14 of which were of the longitudinal type, 3 were transverse, 3 involving the mastoid tip alone, 1 avulsion of the petrous tip and 2 compound fractures. It is to be remembered that fine microscopic fracture lines may be missed in a macroscopic examination and that these fractures may involve the bony labyrinth. As noted by Stenger, the membranous labyrinth may be damaged by the violent disturbance caused by trauma to the head even though the temporal bone is not fractured.

Signs and Symptoms: Some of the most common signs and symptoms of fracture at the skull base involving the temporal bone are: *1.* escape of blood or cerebrospinal fluid from the ear canal or eustachian tube, *2.* vertigo, *3.* diminished or absent hearing, *4.* nystagmus and *5.* headache. The loss of hearing may be slight or marked and of middle or inner ear type. Shemley calls attention to the fact that with irritative lesions of the labyrinth and with the patient conscious there is a nystagmus toward the affected side but if the patient is unconscious there is a conjugate deviation of the eyes directed away from the affected side since the cortical component of the nystagmus is absent. In destructive lesions of the labyrinth the above findings are reversed. In addition to the above symptoms there may be neurological symptoms such as facial paralysis or paralysis of the sixth nerve such as occurred in two of the cases here presented. Due to the presence of infection and an open pathway to the meninges in the cases presented caloric irrigations were not performed.

The signs and symptoms of acute mastoiditis developing after fracture of the skull do not differ from those found in ordinary acute mastoiditis except that facial paralysis and the signs and symptoms of labyrinthine and cochlear involvement occur more frequently. The complications of acute mastoiditis occur more frequently in the cases following skull fracture. This is demonstrated by the cases which are being presented which developed one or more of the following complications: erysipelas, extradural abscess, perisinus abscess, lateral sinus thrombosis, meningitis and brain abscess.

Case 1: J. S., a white male, age 53 years, married, was admitted to Dr. W. Bower's service at St. Luke's Hospital, July 3, 1930, complaining of pain in the right ear and right side of the head. Twenty days previously he had been struck by a taxicab and knocked to the pavement. At that time he bled from the right ear and was taken to a hospital where an X-ray was taken and a diagnosis of fracture of the skull was made. Two weeks later he was discharged as cured. However, since the time of the accident, he had suffered continuously from pain in the right side of the head and the right ear. He also

had vomited several times and had had attacks of dizziness and had been irrational at times. Twenty-three years previously the patient had had a fractured skull and had been unconscious for nine days. One year later, an osteomyelitis of the left ankle had been operated upon resulting in recovery. The family and past histories were otherwise irrelevant.

Examination revealed a middle-aged white male who cooperated poorly and while he responded to painful stimuli he did not respond to questions. The rectal temperature was 105°F., pulse was 124, respiration was 28. There was a slight tenderness in the sub-occipital region and there was marked tenderness with slight edema over

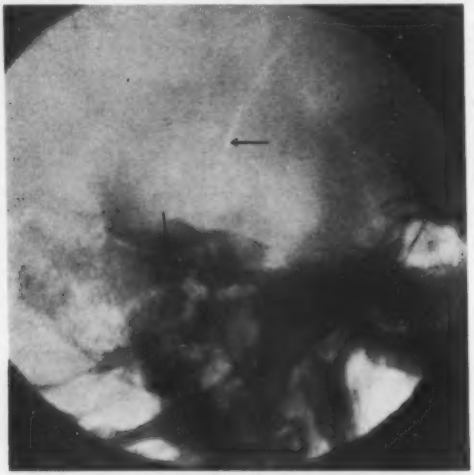

Fig. 1.

the right mastoid process. The superior canal wall was found to be drooping almost to the floor. A profuse, odorless, yellow, purulent, pulsating discharge was present in the canal. The drum could not be seen, due to the narrowing of the canal. The hearing could not be tested. There was a slight edema of both optic nerve heads and a small flame shaped hemorrhage just above the disc in the right fundus. There was slight tenderness and rigidity upon pressure in the right upper quadrant of the abdomen. The Kernig's reaction was questionably present upon both sides. There was a scar over the anterior aspect of the lower left tibia.

Spinal tap showed 30 m.m. of mercury pressure and a slightly cloudy fluid. The butyric was negative; there were 2,250 cells per

cubic m.m. with 96 per cent polymorphonuclear and 40 per cent lymphocytes. The smear was negative for organisms. X-ray showed the left mastoid process to be large, cellular and clear. On the right there was marked infiltration in the zygomatic region and opposite the knee of the lateral sinus. The tip cells were distinctly made out. The appearance suggested acute mastoiditis, most marked in the zygomatic region but with no evidence of softening. There was a vertical linear fracture line about two inches long in the right temporo-parietol region above the external canal. Another fracture line one inch long extended horizontally backward and upward from the junction of the superior and posterior bony canal walls.

A diagnosis was made of fractured skull involving the right mastoid, middle ear and middle cranial fossa with acute mastoiditis and meningitis probably localized.

Operation was performed the evening of admission under nitrous oxide anesthesia. The large cellular mastoid was full of pus and granulations and there was considerable breaking down of the cell partitions. A fracture line extended forward from the parieto mastoid suture horizontally and slightly downward into the superior part of the bony canal and then passed upward through the roof of the middle ear and into the middle cranial fossa. After exenterating most of the mastoid cells a loose piece of bone which proved to be a sequestrum was removed from the floor of the middle fossa. The dura over the base of the temporosphenoidal lobe was covered with granulations and bathed in pus. The posterior canal wall was lowered somewhat. The dura was exposed in all directions until healthy and normal appearing dura was encountered. Upon palpation there was a slight sense of resistance beneath the diseased dura over the temporosphenoidal lobe. This was incised. Two adjacent abscesses which seemed fairly well walled off were discovered just beneath the cerebral cortex. Culture from the abscesses and the mastoid showed staphylococcus and streptococcus hemolyticus. The larger of these was the size of a hickory nut. Drainage was secured by two rubber dam cigarette drains, which extended to the bottom of the abscess cavities, a depth of about one and one fourth inches. The wound was packed with iodoform gauze and left open.

The patient's temperature remained elevated and he was irrational, incontinent and at times delirious. On July 5, the second day post operative, spinal tap showed 24 m.m. of mercury pressure, 17,000 cells with 86 per cent polymorphonuclears and 14 per cent lymphocytes. The smear was negative for organisms but the culture showed hemolytic streptococci and staphylococci. The patient rapidly devel-

oped signs of generalized meningitis and died the morning of the fourth day after operation.

Case 2: R. M., a white male, age 17 years, single, was transferred to the surgical service at Kings' County Hospital on January 22, 1931. He had been admitted to another hospital five days previously in semi-stupor, bleeding from the nose and right ear.

He had been thrown from a rapidly moving motorcycle and had struck his head upon the pavement. He had been irrational and maniacal since admission. X-ray had shown a fracture of the right skull base. Family and past history were not significant.

Examination revealed a well developed young white male, poorly oriented, irrational and very noisy at times. His temperature was 99, pulse 68, respirations 20. Blood pressure 152/60. His neck was rigid. There was ecchymosis around the right eye and both pupils were dilated and reacted sluggishly to light. There was slight blurring of the edges of the optic discs. There was a complete right facial paralysis. Battle's sign was present on the right and there was a bloody discharge in the canal of the right ear. Knee jerks were absent.

Spinal tap showed slightly increased pressure and blood tinged fluid which was sterile. Urinalysis, blood and spinal Wassermann's were negative. X-ray of the skull showed a fracture line extending upward and backward in the right occipital bone for a distance of about 4 cm. from the region of the knee of the right lateral sinus.

Three days after admission slight weakness of the left external rectus was noted and the following day there was complete paralysis. Ten days after his injury, the patient became more rational and began to take fluids by mouth. On the sixteenth day of his illness he first complained of pain in the right ear and it was noted that the discharge was purulent. His facial paralysis was less marked.

Otological examination on February 3, revealed displacement of the right auricle anteriorly and laterally and there was a red fluctuant tender swelling over the right mastoid with slight edema of the surrounding scalp. The canal was filled with thin, odorless, flaky pus and the superior canal wall was sagging nearly to the floor so that the drum could not be visualized. There was no nystagmus. Conversational voice was heard at three feet in the right ear. There was moderate right facial weakness and paresis of the left external rectus. The temperature was normal. There were 14,600 leucocytes per cubic m.m. with 83 per cent polymorphonuclears, small lymphocytes 4 per cent, large lymphocytes 5 per cent and transitionals 8 per cent, 4,710,000 red blood cells per cubic m.m. It was thought

that the patient had acute suppurative mastoiditis and a subperiosteal abscess secondary to a fractured skull.

X-ray of the mastoids showed the fracture of the sinus plate above noted. Both mastoids were of moderate size, of pneumatic type with increased density throughout the right mastoid indicating the presence of inflammatory products, but there was no evidence of destruction.

Operation was scheduled for 4:00 P. M. of the following day and at 12:00 noon the temperature rose to 102 degrees but there was no accompanying chill. At operation a subperiosteal abscess containing about 10 c.c. of pus was found overlying a fracture line which extended horizontally backward and slightly upward from the junction of the posterior and superior bony canal walls and extended through the sinus plate obliquely in the region of the knee. At its

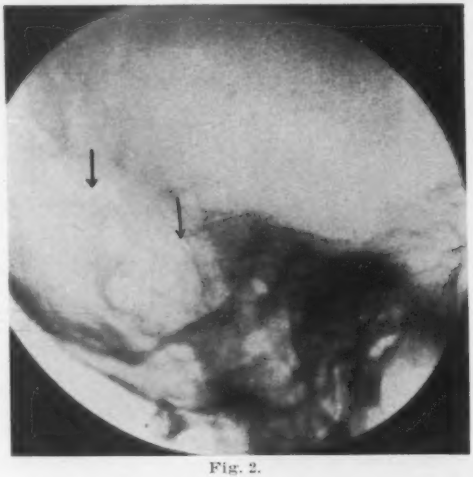

Fig. 2.

anterior partition the fracture line passed upward and apparently extended forward into the middle cranial fossa. The entire mastoid contained pus but there was little softening. There was a perisinus abscess at the knee, also slightly below and posterior to it with luxuriant granulations. The sinus wall was thickened. There was no demonstrable thrombus and pulsation was present. The sinus was exposed nearly to the bulb and backward for about one inch so that healthy appearing sinus wall was uncovered in both directions. The adjacent dura of the posterior and middle fossa appeared normal. The sinus was not opened. After completing the simple mastoid operation, the wound was packed with iodoform gauze and left open. Culture of the mastoid wound showed nonhemolytic streptococcus.

The following day the temperature rose to 102.4. The general condition was unchanged. Blood culture was negative. On the second day postoperative the temperature rose to 108.4 (rectal) preceded by a chill, and blood culture showed nonhemolytic streptococci. A 500 c.c. blood transfusion was given. Jugular ligation was advised but consent for operation was withheld by the family until the following day. Under local anesthesia the carotid sheath was exposed from the clavicle below nearly to the mastoid tip, above. The wall of the jugular vein was thickened in the upper portion but no thrombus was demonstrated. Ligation was performed just above the facial vein and the facial vein was also ligated. The jugular vein was ligated again below the middle thyroid vein. Under light nitrous oxide anesthesia the sinus was then opened and a thrombus found in the region

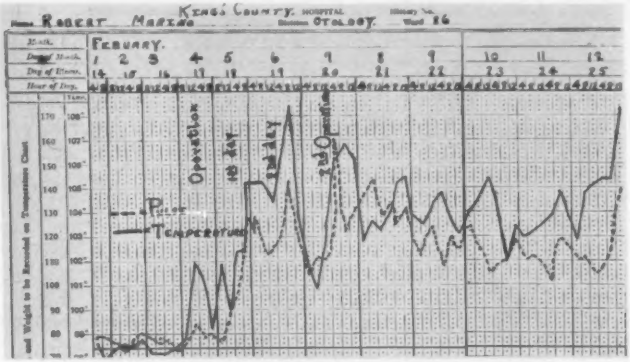

Fig. 3.

of the knee. The incision through the lateral sinus wall was extended backward for one inch and free bleeding obtained. The sinus was occluded by a small plug of iodoform gauze and the wound repacked.

In spite of repeated transfusions and suppurative treatment the temperature remained elevated and the patient developed metastatic abscesses of the right ankle and foot and the left wrist. Death occurred on February 12, nine days after the first operation. Autopsy performed by Dr. Hala revealed a fracture 2 c.c. in length just beneath the lambdoid suture in the occipital bone. There was also a fracture extending from a point about three quarters of an inch to the right of the right posterior clinoid procesa of the sphenoid bone through the petrous portion of the temporal bone parallel to its anterior border for one inch. This crossed the facial canal at the knee and extended into the tegmen tympani. There was no evidence of fracture of the bony labyrinth. The cerebrum showed marked con-

gestion and edema and the subpial vessels were markedly injected. There was a softened area on the inferior surfaces of both frontal lobes extending from the anterior poles backward to the termini. There was no evidence of meningitis. The right transverse sinus in its anterior portion was collapsed and its lateral wall had been incised. There was an infected clot in the posterior portion of the transverse sinus and the upper portion of the jugular vein just below the jugular bulb also contained free pus. The incision in the neck contained pus. There was moderate adiposis of the myocardium and acute broncho pneumonia of both lungs. There was a localized, subcutaneous abscess the size of the palm of the hand on the dorsal aspect of the right foot and ankle and another smaller subcutaneous abscess over the dorsal aspect of the left wrist and hand.

Case 3: A. S., a white male, age 59 years, single, was transferred to Kings' County Hospital on April 24, 1931, from another hospital. He complained of pain in the right frontal region and right ear and discharge from the ear. The family and past histories were negative except that, since the age of 25, the patient had been subject to epileptic seizures with generalized convulsions and loss of consciousness. The previous ear history was negative.

During an epileptic seizure on April 10, he fell to the sidewalk and struck the right side of his head. He regained consciousness in a few minutes and was taken by ambulance to the hospital where examination revealed that he was rational and mentally clear. There was a hematoma and a laceration of the scalp in the right temporal region and a perforation of the right drum membrane with discharge of clear serous fluid in the canal. He was kept in bed and no other treatment instituted. The only complaint was pain in the right side of the head.

Examination upon admission at Kings' County Hospital revealed an elderly white male acutely ill with a temperature of 101, pulse 104, respirations 28. Anteriorly, posteriorly and above the right ear was an indurated tender area about 10 to 15 cm. in diameter and the auricle was red, swollen and tender upon manipulation. There was a copious discharge of greenish yellow, slightly foul pus from the canal. The Babinski was equivocal. The blood Wassermann was negative. There were 8000 white blood cells per cubic m.m., polymorphonuclears 65 per cent, lymphocytes 32 per cent, transitionals 2 per cent, eosinophiles 1 per cent, hemoglobin 78 per cent with 3,872,000 red blood cells per cubic m.m. Blood sugar was 177 mgs. per 100 c.c. The urine showed only a faint trace of albumin with an occasional hyoline cast and leucocyte in the sediment. Culture from the right ear showed nonhemolytic streptococci.

It was thought that the patient had erysipelas and 20 c.c. of erysipelas antitoxin was given and an alum acetate dressing applied to the right side of the head and right ear. A low carbohydrate diet was prescribed. Two days later the discomfort was less and the edema and swelling were slightly diminished but the discharge from the ear did not decrease. The blood chemistry was normal.

The patient was first seen by the ear service 18 days after his injury. At that time the temperature was normal. The auricle was red and swollen and there was an indurated, reddened, tender swelling anteriorly, posteriorly and above the auricle. There was moderate deep tenderness over the mastoid tip and a profuse, purulent, slightly

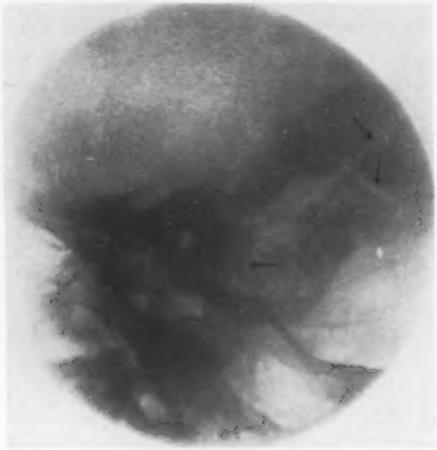

Fig. 4.

foul discharge from the canal. The superior canal wall was sagging nearly to the floor and the drum was not seen. Conversational voice was heard at two and a half feet in the right ear. There was slight horizontal nystagmus to the right upon looking to the extreme right.

X-ray showed that both mastoids were of moderate size, pneumatic type, with a marked decrease of illumination throughout the cells of the right mastoid with moderate destruction. There was a fracture line through the sinus plate in the region of the knee extending upward and backward into the parietol bone: a second line of fracture extended downward and posteriorly from the occipitomastoid suture line for one inch.

On the following day, operation was performed under avertin anesthesia. A subperiosteal abscess containing several drachms of pus was found. There was a fracture through the sinus plate at the

knee which extended backward and upward diagonally across the horizontal part of the lateral sinus to the region of the asterion. At this point it branched and an upper limb passed upward and backward into the parietal bone for about one and one half inches and a lower branch passed downward and backward just posterior to the occipitomastoid suture line. At the knee of the sinus the fracture line passed downward anteriorly across the mastoid process ending about one quarter to one half inch above the tip so that the tip of the mastoid process was lifted out in one piece. Another fracture line passed horizontally anteriorly and downward from the knee of the sinus through the juncture of the posterior and superior bony canal walls. The mastoid cells were broken down and contained pus. After most of the mastoid cells had been exenterated the posterior canal wall was lowered and part of the epitympanum and zygomatic cells were removed. Over the horizontal part of the lateral sinus midway between the knee and the torcula a perisinus abscess containing about two drachms of pus was found. The external sinus wall in this region was covered with granulations. There was no evidence of thrombosis of the sinus. The surrounding dura of the posterior and middle fossa was discolored. In order to expose normal dura in all directions it was necessary to remove bone above and below the fracture line over an area about one by two inches. An accidental nick of the sinus caused free bleeding from both directions. This was controlled by plugs. The wound was left open and packed with iodoform gauze. Culture from the mastoid wound showed nonhemolytic streptococci.

After operation, the temperature remained elevated to from 102.4 to 104.6, there was considerable discharge from the wound. The erysipelas in the wound area flared up and spread forward over the right side of the face. On the second day 20 c.c. of erysipelas antitoxin was given. On the third day post operative the patient rapidly developed signs of generalized meningitis and died the morning of the following day.

Autopsy performed by Dr. Martin, on May 4, revealed that a mastoid operation had been performed on the right side and that the bone posterior to the mastoid over the region of the transverse sinus and above and below for an area of about one by two inches had been removed. The outer wall of the sinus was thickened and covered by granulations in its mid-portion and there was a small incision in this area. There was no organized or infected clot in the sinus. There was no evidence of subdural hemorrhage. The brain showed a diffuse, purulent exudate over the right cerebral hemisphere, this was also present at the base of the brain, covering both lobes of the cere-

bellum on its under surface and the pons. Upon stripping the dura a fracture line was found passing through the petrous portion of the temporal bone and extending laterally from the foramen lacerum in the right middle cranial fossa through the roof of the carotid canal passing just anterior to the labyrinth and the genu of the facial nerve. At the lateral extremity the fracture line passed backward through the roof of the middle ear but the medial wall of the middle ear was not involved.

CONCLUSIONS.

1. Three cases of acute mastoiditis developing after basal skull fracture are presented. All terminated fatally.

2. Complications were found in all three cases. In case 1 there was an extradural abscess and two brain abscesses. Case 2 had a subperiosteal abscess, perisinus abscess, and sinus thrombosis. Case 3 had erysipelas, subperiosteal abscess, perisinus, and extradural abscesses.

3. The cause of death was meningitis in two of the cases and septicemia in the other one.

4. The mortality is high and complications are common due principally to the presence of infection with an open pathway to the meninges. The best chance for success would seem to be in early diagnosis and early surgical intervention.

5. Acute mastoiditis is a delayed complication of basal fractures and the signs and symptoms are similar to those of acute mastoiditis from other causes.

6. Patients with head injuries should have an ear examination as soon after the time of the injury as possible, and the ears should be closely watched thereafter.

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AGRANULOCYTIC ANGINA. REPORT OF FOUR CASES.

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Passaic, N. J.

Every busy practitioner has met cases in his practice which at first inspection did not differ from an ordinary sore throat, diphtheria, Vincent's angina or peritonsillar abscess.

The doctor administers diphtheria serum or, in cases of spirillum and fusiform infection, gives arsenicals, eventually incises a peritonsillar swelling and usually promises healing after a few days to the anxious family.

But the promised few days have gone, the condition does not improve; on the contrary, the prostration of the patient grows rapidly worse and, much to the surprise of the doctor and family, the patient dies.

The very disagreeable question, "Why?" is answered by the doctor with the common phrase, "an unusually bad septic sore throat." The family accepts the diagnosis but the doctor despairingly acknowledges to himself that he just has given another name for his ignorance.

This ignorance is no longer permissible. A certain group of these fatal throat cases became a pretty well defined entity, described by Schultz and Werner in 1922 as agranulocytic angina. Their definition of the disease was, an acute disease with high fever, necrotic processes in the oropharyngeal tract and characteristic changes in the blood. There is a marked leukopenia, the number of the white blood cells being 1,200 or less, and there is a very marked agranulocytosis, 20 per cent to *nil*.

Absence of hemorrhagic diathesis, icterus and rapid fatal course was characteristic in his published cases. These later features are, according to Schultz's later communications, not always present in all cases of agranulocytic angina.

The first communications about the disease are only a few years old and there are only relatively few cases published in the literature. This is probably the reason that we know practically nothing positive about etiology and therapy of this disease and that is the reason that even the characteristic definition of the sickness has undergone some changes in the last few years.

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To purify the definition and the chief characteristic of the disease we hereby take the liberty to recount three cases of our own treatment and one case which we saw in consultation.

Case 1: M. D., middle aged woman, very intelligent, and in the beginning of the disease very responsive. Family history insignificant. Three years ago had an acute attack of right frontal sinusitis, which subsided on conservative treatment. But she suffers sometimes with occasional right-sided frontal headaches. Several years ago she suffered with slight infiltration of the apex of the right lung. She did spit blood that time and the sputum was positive for Tbc. But in the last five years she had no trouble with her lungs. Since her childhood she always had some ailment in her abdomen. Five years ago they removed an ovarian cyst from her abdomen.

Several months ago she started to suffer with diarrhea and cramps. She came in the hospital to have a thorough laboratory once-over of her feces, blood, etc. By going over the routine laboratory tests Dr. Kastler found that most of the tests were normal or nearly normal, except the blood picture, which showed the following: Red blood cells, 4,200,000; white blood cells, 2,000; polymorphonuclear neutrophils, 0 per cent; large lymphocytes, 35 per cent; small lymphocytes, 65 per cent.

I was called to the patient, who at first sight impressed me as a light case of some chronic disease. All her complaints were concerned about her abdomen. She did not complain about her throat at all on the first day. Temperature was 101°, pulse 82. Examination of her nose showed some hypertrophy of the middle turbinate, right. The mucous membrane of the throat and mouth was pale but I could visualize on the upper pole of the right tonsil, in a cryptic cavity, a dirty gray necrotic exudate, the size of a pea. The environment of the tonsil, the anterior and posterior pillar was curiously neither swollen nor red.

The next day she started to complain of severe sore throat. The temperature increased to 103°, pulse 86, and she became at once very weak. On the upper pole of both tonsils we could now see a dirty gray exudate the size of a bean. We treated her throat with Fowler solution and she got neosalvarsan intravenously.

Blood count: Leukocytes, 1,000; polymorphonuclear neutrophils, 0 per cent; large lymphocytes, 40 per cent; small lymphocytes, 60 per cent. Temperature 106°, emesis often, prostration. Blood transfusion given.

Aug. 24: Temperature 104°, pulse 100. She does not take nourishment. The cavities in the tonsils grow and the necrosis on right

side destroys parts of the anterior pillar, too. Leukocytes, 600; no polymorphonuclear neutrophils, 70 per cent small lymphocytes, 30 per cent large lymphocytes.

Aug. 25: Temperature 104° One necrotic spot on the left anterior pillar, too.

Aug. 26: The temperature continues to be 104°. The place where the neosalvarsan injection was given is very red and swollen. Red cells, 3,900,000; leukocytes, 100; small lymphocytes, 85 per cent; large lymphocytes, 15 per cent; polymorphonuclear neutrophils, 0 per cent.

Aug. 28: Patient only takes few sips of water. Temperature rises to 105°, pulse 130.

Aug. 29: Patient dies, with final rise of the temperature, 106.4°.

There are several very interesting features in this case. First of all, the case started before our eyes, because Dr. Kastler, who examined the patient a few days before the admittance, did not find any evidence of the change of the blood picture at that time. The first sign was the characteristic blood count. The patient did not complain about sore throat until next day. We may state assuredly that the change of the blood was at least simultaneous with the throat trouble, very probably even somewhat previous to the latter; secondly, the daily-taken blood counts show that the amount of the leukocytes diminished daily, the polymorphonuclear neutrophils disappeared and did not come back in the picture.

The second case is a strong young man, C. W., in the twenties. He never was sick before. There is no hereditary sickness or disposition in his family. On May 1, 1930, he started to develop a sore throat and went to a university clinic. The diagnosis was necrotic sore throat. His temperature on the first day was 101° and his blood picture, taken in the university, the following: Red blood count, 4,500,000; white blood count, 14,200; hemoglobin, 90 per cent; neutrophils, 80 per cent; lymphocytes, 17 per cent; large monoculears, 3 per cent. In the university they treated him for three days. Day after day he became worse and worse. He left the university and went home and was treated there for ten days. He had continuously high fever, but no chills. His throat was very sore, especially on the right side.

May 12: He came in the hospital; his temperature was 105°, pulse 120. Patient is responsive and intelligent. His mind is clear. His lips are cracked, tongue coated. We can see on both tonsils a dirty grayish exudate, which is especially extensive on the right. The anterior part of the right side of the nasal septum shows some dilated

veins, which bleed easily. Dr. Martland examined his blood picture and made the diagnosis of agranulocytic angina. Red blood count, 4,600,000; white blood count, 300; neutrophils, 1 per cent; small lymphocytes, 99 per cent. He gets 0.45 neosalvarsan and 300 c.c. blood transfusion. He vomited a few times.

May 13: His temperature is continuously high, 104-105°. Pulse is a little better, 110-120. He is comfortable but he cannot sleep nights, and vomited once. We give him a mouth wash and we paint his throat with mercurochrome.

May 14: Patient is restless, does not complain, but his temperature is still 104-105°, his pulse 110-120. His right arm starts to swell at the place where the neosalvarsan injection was made.

May 15: Temperature had a slight morning remission to 103.6° and subsequent rise to 106°. Patient's mouth odor is fetid. He is nauseated and vomits; swelling on arm grows. White blood cells, 2,600; polymorphonuclear neutrophils, 2 per cent; small lymphocytes, 97 per cent; large lymphocytes, 1 per cent.

May 16: The fifth day in the hospital and eighteenth day of the sickness. The temperature goes down in the form of lysis. The morning temperature is 103°, the afternoon temperature is 104.4°. The patient is not so restless, feeds better and has more sleep, but the swelling on his right arm grows.

May 17: The patient's pulse is weaker, the patient is listless, but the morning temperature is 102°, the afternoon temperature 103.4°. White blood cells, 1,000; polymorphonuclear neutrophils, 4 per cent; small lymphocytes, 90 per cent; large lymphocytes, 6 per cent.

May 18: Decidedly better today. Lowest temperature 100.4°, highest temperature 103°, pulse better, patient brighter, arm painful and swollen.

May 19: Highest temperature 102°; lowest temperature 100°; 300 c.c. blood transfusion given by Dr. Whelan.

May 20: Highest temperature 100°, lowest temperature 98.8°. Red blood count, 5,400,000; white blood count, 5,000; hemoglobin estimate, 95 per cent; polymorphonuclear neutrophils, 45 per cent; small lymphocytes, 50 per cent; large lymphocytes, 5 per cent.

May 21: White blood count, 6,400; polymorphonuclear neutrophils, 55 per cent; large lymphocytes, 40 per cent; young forms myleocytes, 5 per cent. Patient improved, throat clearer.

May 22: White cells, 7,300; polymorphonuclear neutrophils, 55 per cent; small lymphocytes, 35 per cent; large lymphocytes, 5 per cent; Young forms myleocytes, 5 per cent. The patient is in wheel chair for one-half hour.

May 22-30: Patient had for a few days 97-98° temperature. Has yet a small necrotic exudate on the right side.

July 27: White blood cells, 8,000; polymorphonuclear neutrophils, 60 per cent; small lymphocytes, 30 per cent; large lymphocytes, 7 per cent; transitionals, 3 per cent.

On May 30 the swelling on the arm was opened. The recovery was uneventful. Blood examination on June 4th was: White blood cells, 10,000; polymorphonuclear neutrophils, 56 per cent; small lymphocytes, 36 per cent; large lymphocytes, 6 per cent; transitionals, 2 per cent.

June 12: White blood cells, 8,200; polymorphonuclear neutrophils, 66 per cent; small lymphocytes, 30 per cent; eosinophils, 2 per cent; basophils, 2 per cent.

June 13: Left the hospital, apparently quite normal and healthy.

Our second case seems to confirm the findings of other authors, that the prognosis in male patients is decidedly better than in females. Very important is the fact that the first blood picture taken in the university clinic is quite normal, with leukocytosis instead of leukopenia, with the throat infection already present. It seems to be sure in this case that the damaging of the hemopoetic system is secondary to the throat infection. In the beginning, contrary to the expectation, the throat infection caused a leukocytosis. The leukopenia appeared six days later. The fall of the temperature was accompanied with marked increase of the neutrophils and large lymphocytes. Young form myelocytes appeared in the blood, too.

Case 3: J. C., 14-year-old girl, complains that she has been feeling weak for several weeks. Two days previously she started to have sore throat and fever. I first saw her on March 27, 1929. The patient made the impression of a very sick girl. She talked with difficulty and every movement of the mouth was very painful. Temperature 103°, pulse 130. Mucous membranes were pale, lips dry, tongue coated. The uvula and the right anterior pillar were edematous. The swelling of the anterior pillar covers the right tonsil so that we cannot see its surface. The left tonsil and its surroundings were intact. My diagnosis was the beginning of a peritonsillar abscess. I was quite sure that I wouldn't find liquid pus behind the edematous pillar yet, so I decided to wait. On March 27 the throat did not change much. The edema was rather smaller. Certainly it did not grow. But the prostration of the girl was worse, the pulse, 130, weak and the temperature 103°.

On the next day's examination the whole picture changed. The edema disappeared. We could now see the left tonsil, whose surface

was covered by a dirty grayish membrane. A similar membrane covered the right anterior pillar, too. I made the diagnosis of necrotic tonsillitis and ordered a laboratory examination for diphtheria and Vincents angina and other germs. The smear showed many staphylococci, streptococci and diphlococci. The twenty-four-hour culture on Loeffler serum showed positive diphtheria bacilli. I consequently gave her 20,000 units of diphtheria serum.

Incidentally, the blood was examined, too, and showed the real character of the disease: Red blood cells, 4,200,000; white blood cells, 1,100; polymorphonuclear neutrophils, 10 per cent; small lymphocytes, 60 per cent; large lymphocytes, 30 per cent. The child was given a transfusion of 300 c.c. citrated blood and transferred into the isolation hospital, under the care of Dr. Ryan.

April 1: The child had temperature 103°, pulse 130. Very weak. Blood count: Red blood cells, 3,900,000; white blood cells, 700; polymorphonuclear neutrophils, 1 per cent; small lymphocytes, 65 per cent; large lymphocytes, 34 per cent.

April 2: Some bleeding from mouth.

April 3: Child expired.

Very interesting in this case is its similarity to peritonsillar abscess, which on the first day did not differ at all from the usual picture of peritonsillar abscess, with the exception that there was no pressure, no fluctuation or pointing to be seen. The edematous tissue was quite flaccid all the time.

The history of the fourth case is very similar. T. T., sick for two weeks with sore throat and fever. She received diphtheria serum and a gargle and her condition became progressively worse and worse, so that she came into the isolation hospital. On admission her temperature was 101°. She was very pale, tongue coated, dry. No bleeding from gums. Left tonsil with uvula, normal. On the place of the right tonsil we see a grayish necrotic tissue. The upper part of the anterior pillar is destroyed; the lower part swollen, rigid and thick. An adjacent small part of the tongue is necrotic and covered by the same dirty, bad smelling exudate. The blood picture was characteristic: Red blood cells, 4,000,000; white blood cells, 700; polymorphonuclear neutrophils, 4 per cent; small lymphocytes, 56 per cent; large lymphocytes, 40 per cent.

March 16: The temperature was elevated to 105°, pulse 140. Small streaks of blood on the right necrotic surface. Very prostrated.
March 17: Temperature 105°, pulse 144; expires.

The *diagnoses* of these four cases were simple. All of them showed the characteristic short duration, the necrotic angina, the extreme

prostration, high fever, characteristic leukopenia and agranulocytosis, with practically no change in the red blood cells.

We see a similar blood picture in aplastic anemia, in kala azar and aleukemic leukemia.

Aplastic anemia does not show such severe and rapid progress; there is usually no such temperature and there is no typical throat condition.

The aleukemic stage of leukemia may show the same changes in the blood but the death does not come so quickly. We are usually able to detect enlargement of the spleen and glands. There is no necrotic angina present.

Kala azar, or visceral leismaniasis is not common in our states. Its usual habitat is India, Algeria, southern Europe. The leukopenia and agranulocytosis is present, but we find in the leukocytes the typical parasite.

ETIOLOGY.

The cases presented until now are mostly from Europe and the temperate part of America.

The female sex appears to be very much more affected than the male.

The age is usually above ten, but in middle age it is more prevalent. The disease appears in every season of the year; there is no proof of the contagiousness of the disease. The cases seem to be sporadic.

There are several theories explaining the cause of the disease.

One theory considers that throat infection is the primary cause. The germ or poison goes through the tonsils into the hemopoetic system and damages it secondarily. Our second case, which we observed from the very beginning, seems to confirm this theory.

According to the second theory the essential features of agranulocytic angina consists in the primary damage of the hemopoetic functions of the bone marrow. The gangrene formation and the phenomena of sepsis are only secondary.

The damage of the hemopoetic system is brought about, according to some authors, by some infectious agent. Others consider some endocrine disturbances as a cause of this damage. Frank observed similar injury of the bone marrow after poisoning with benzol or salvarsan.

According to a third version of this theory, agranulocytic angina can only develop in persons with hypoplastic bone marrow. This last version is contradicted by cases which heal.

The endocrine theory is assisted very much by the fact that the overwhelming majority of the cases belong to the female sex.

There are a few authors who do not consider agranulocytic angina as a clinical entity at all, but as a terminal condition of many diseases.

The sickness was usually ushered in by some *prodromal* symptoms, which seem to be weakness, fatigue, loss of appetite, headaches, sometimes sore throat. The duration of the sickness in our cases were ten days to two weeks. After this time it ended with death or with recovery. The recovery in our case seemed to be perfect, but the blood picture showed some young forms long after the temperature became normal.

Temperature was very regular in our four cases. It never started with a chill, but in two cases we observed from the beginning it ascended gradually to high temperature, 104-105°, and kept on continuously as febris continua without larger remission until death. Very often with hyperpyrexia, 106° and above, before death. In the case of recovery the temperature did not fall by crisis, but slowly by lysis. The defervescence in our last case lasted four days. It looks very much like the typical curve of typhoid fever.

Digestive System: In the height of the disease with high temperature there is a loss of appetite. The pain in the throat is considerable and hinders swallowing.

The chief pathological changes are on the tonsils, which are partially or totally covered by a grayish necrotic exudate. The necrotic process is sometimes very extensive. In one of our cases it extended to the pillars, tongue, bucca, and sometimes gums. In our third case it destroyed the tonsils totally. We had in two cases bleedings from the necrotic area on the tonsils, probably caused by the necrosis of some vessel.

There was in all our cases considerable constipation. In no case were there enlarged gland formations on the neck or anywhere else.

Organs of Respiration: In not one of our cases did the lungs participate in the process. There was no cough, no pain in the chest, no consolidation of the lungs, no pleurisy.

There was no hoarseness, no laryngitis, no coryza, but occasional nose bleeds of short duration.

The *nervous system* seems to be affected from the beginning. The patients were all extremely prostrated. As the disease progresses their reaction to questions is slow; in the final stage they become dull, incoherent, delirious.

The *organs of circulation* show no characteristic changes. There is no murmur, no dilation of the heart. The pulse follows the temperature and usually is satisfactorily full until two or three days before death. The blood shows the characteristic leukopenia with

the absolute diminishing of the white blood cells to 700 and the neutrophils to 0.5 per cent, with relative increase of the lymphocytes. The red blood cells and the hemoglobin are not very much changed. During recovery we found young form myelocytes in the blood.

Very characteristic is the diminished resistance of the body to neosalvarsan injections. In two cases of ours, the injections were followed by swelling at the place of injection, notwithstanding that they were made by very competent men. In the case which recovered this swelling turned to suppuration and had to be opened.

In not one of our cases were the *bones, joints* or *muscles* affected in any special way.

The *uropoictic* system was hardly affected. The urine was sparse but contained neither albumin nor blood sugar. The laboratory examination found in one case hyalin casts.

Prognosis: Out of our four cases only one is living. Other authors with larger number of cases average 10-15 per cent. It looks like the prognosis of male patients is much better than female patients.

Therapy: We used blood transfusion, neosalvarsan, mouth wash, antiseptic treatment of the necrotic area of the tonsils. But we are convinced that all this therapy but slightly influences the fate of our patients.

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195 Lexington Avenue.

CELLULITIS OF PHARYNGEAL TISSUES—MEDIASTINITIS: DEATH.

Dr. Harold M. Hays, New York.

On Dec. 11, 1930, Miss E. L. L., age 25 years, consulted me, complaining of a severe sore throat which she had had for a few days. Examination showed considerable swelling of the tissues surrounding the right tonsil and extending upward to the superior tonsillar fossa. A tentative diagnosis of a peritonsillar abscess was made.

On the following day the condition was found to be worse and there was considerable dysphagia. Temperature was 101°. She was immediately sent to the Park West Hospital. She was taken to the operating room, where, under ethyl chlorid anesthesia, a more careful examination was made. The mucous membranes of the throat, particularly on the right side, were found to be edematous, the edema extending along the right pharyngeal wall to the larynx. Numerous incisions were made above the tonsil, into the tonsil and in the edematous area behind the tonsil but no pus was encountered.

The patient was put back to bed and placed on the usual routine treatment—hot throat irrigations, mercurochrome drops in nose to nasopharynx, icebag to the neck and antipyretics. In spite of the fact that the throat condition cleared up in a few days, the patient looked severely ill and the temperature, instead of returning to normal, ran an irregular course between 102° and 104°. Blood examination showed 11,000 white blood cells with 85 per cent of polymorphonuclears. Blood cultures were negative on three tests. Examination of the urine showed a slight amount of pus, indicative of a possible pyelitis.

On the fourth day the patient complained of severe pain on the right side of the neck and a tender swelling appeared in the region of the right lobe of the thyroid. We sincerely hoped that a localized infection would present itself which could readily be cared for but this soon disappeared to be followed by a similar swelling on the left side which, to a certain extent, remained until death. On the sixth day the patient had a chill and the temperature rose to 106°. From that day on, for a period of over two weeks, irregular temperatures accompanied by chills occurred.

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On Dec. 20 Dr. Victor Meltzer was called in and took care of the case. Rales could be heard at the base of the left chest and a tentative diagnosis of a deep-seated bronchopneumonia of the left lung was made. This was later corroborated by physical signs and X-ray pictures. In spite of every known therapeutic measure, no relief of the symptoms occurred. The abdomen became rigid, pain in the lower chest became intense and a sub-phrenic abscess was suspected. On Dec. 29 Dr. Harlow Brooks was called in consultation and corroborated the findings of Dr. Meltzer. By that time well developed physical signs of bronchopneumonia were present on the left side and slight evidence of a similar condition could be found at the right lower base. A needle had been inserted into the left pleura but no fluid or pus was found.

The patient's condition became decidedly worse. There was almost complete loss of voice so that she could speak only in a whisper and there was considerable difficulty in swallowing. Fluid would pass to a certain part of the esophagus and either be regurgitated or go down slowly. We were at a loss to account for these symptoms at the time.

On New Year's Day it was considered advisable to call in a thoracic surgeon and Dr. Harold Neuhof consulted with Dr. Meltzer and myself. He found no other physical signs than those enumerated above but considered the late symptoms, aphonia and dysphagia, of great significance, suggesting pressure in the mediastinum. He inserted a needle into the chest at about the eighth dorsal vertebra. There was an immediate withdrawal of a considerable quantity of foul smelling pus, probably anaerobic in character.

The patient was immediately taken to the operating room, where, under local anesthesia, the seventh and eighth ribs were resected and a direct opening made into the mediastinum. The cavity was considerably filled with dirty, straw-colored fluid pus and fibrinous exudate, which formed a number of pockets in which pus was lodged. This fibrin was cleaned out and the pus withdrawn with suction until a clean cavity remained, which was packed with iodoform gauze. The pleura looked normal and there seemed to be no evidence of pleural effusion.

The patient did not react well from the operation and died at eleven o'clock the next morning.

It is regrettable that more cases of this kind are not reported. Throat infections, of a severe type, even after the tonsils have been removed, are frequently seen and the sequellae are most disastrous. It is seldom that a mediastinal infection is thought of although the

pathway of infection from the throat is most obvious. In many such cases, frank pus in the throat can not be demonstrated. The process is a cellulitis of the posterior pillar of the fauces and the pharyngeal wall. In the majority of cases, extensive incisions, under ethyl chlorid general anesthesia will clear up the trouble. But extension may occur. However, this has been the first case in which I have seen extension to the mediastinum with demonstration of free pus in this closed thoracic cavity.

64 East 58th Street.

DILATORS FOR THE PHARYNGEAL ORIFICE OF THE EUSTACHIAN TUBE.*

DR. SIDNEY YANKAUER, New York.

Swelling of the mucous membrane surrounding the pharyngeal orifice of the Eustachian tube is one of the most familiar pathological changes in chronic catarrhal otitis, and one of the earliest taught to students. But that the changes which take place here are such that they could properly be described as "strictures" is not so clearly appreciated. Actual examinations demonstrate that whereas the normal pharyngeal orifice permits the passage of a bulbous instrument about 7 m.m. in diameter, yet in extreme cases of catarrhal otitis, the opening may be contracted to less than 2 m.m. in diameter—almost a pin-point opening. That such a strictured orifice cannot be dilated by blowing air through it, or that the infiltrated mucous membrane which forms its walls cannot be melted away by a few applications of mildly astringent solutions must be clear, upon consideration of the nature of the condition. The only hope of relief must come from mechanical measures. For this purpose dilators are provided, consisting of a suitable shank, with a conical head on one end. The small end of the cone is small enough to engage an opening of

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2 m.m. diameter, the large diameter of the cone being 5, 6, 7 and 8 m.m., respectively.

When the dilator is to be passed, the pharyngeal orifice of the tube is exposed by passing the direct nasopharyngeal speculum. The dilator is introduced into the orifice and the entire conical end is passed through it. A larger one is then substituted, using one too large to pass entirely into the tube. It is held in place while the speculum is removed by passing it over the shank of the dilator. The latter is held *in situ* for a number of minutes, and after its removal the speculum is again passed and an application of 50 per cent argyrol is made to and into the dilated orifice. Or, the dilator may be removed after a few minutes, and the application made at once.

In one case in which a web-like structure was found, incision would have been indicated, but in fact, the diagnosis was not made until the web was ruptured by the passage of the dilating instruments.

The use of diathermy for incising these structures has not been attempted by the author.

The introduction of the direct nasopharyngeal speculum, followed by dilators, requires thorough cocainization of the nasopharynx, the soft palate and the pillars of the fauces. For this reason I have tried to distinguish those cases in which the use of these instruments must be considered indispensable, rather than merely desirable. Practically, this means cases in which the constriction is extreme. While there seem to be no symptoms which are positively characteristic of extreme contraction, there are a few conditions which are sufficiently suggestive for practical purposes. First, the cases in which catheterization is difficult. That is, regardless of which set of rules for passing the catheter is followed, it is difficult to bring the catheter exactly opposite the tubal opening. Merely the fact that the catheter cannot be readily passed without using the nasopharyngoscope in the other nostril is suggestive of a very small tubal opening.

Second, cases in which Politzerization does not produce a satisfactory blowing sound in the auscultation tube characterizes a small tubal opening, provided stricture at the isthmus has been previously excluded.

When the Politzer bag is used for this purpose, the character of the sound heard in the auscultation tube depends also upon the force used in compressing the bag. This varies considerably with the surgeon using the bag. In my own practice, Politzerization is not performed by hand, but compressed air is used through a suitable nasal tip. This permits me to measure the air pressure that is used. With the apparatus set up as used by me, when five pounds of air or more

is necessary, the tubal orifice is considered to be excessively contracted.

Third, whenever all methods fail to improve the hearing, the tubal orifice is measured by passing these instruments.

While these indications are highly suggestive of extreme stenosis, I believe that no diagnosis of the physical condition of the ear in chronic deafness is complete without concrete knowledge, gained from actual measurement, of the degree of constriction of the pharyngeal orifice of the Eustachian tube.

When the author first presented his nasopharyngeal speculum, there appeared at the same time another method of direct examination of the nasopharynx by Cyergaie, of Budapest. In this method the patient was placed on his back, with his head overhanging the table. Secretions from the nose and the pharynx constantly bathed the orifice of the tube, so that continuous suction had to be used. The instruments for dilating the tube were numerous, difficult to make, and to use. By the author's method the patient is upright, and the secretions flow away from the tubal orifice. The instruments used are fewer and simpler, and the entire procedure has been made suitable for ambulatory practice.

The reaction from passing the dilators is very slight, and the procedure may be repeated at short intervals, until the necessary amount of dilation has been accomplished.

Following dilation of the pharyngeal orifice of the tube, catheterization becomes easier and Politzerization easier and more efficient, and the treatment of the otitis accelerated. It may be especially noteworthy that the patients have volunteered the information that the "cloudiness" of the ear diminishes and that the tinnitus is lessened after each treatment.

Experience has convinced me that the addition of this procedure to our usual methods of treating catarrhal otitis is well worthy of more general employment.

These dilators are manufactured by George Tiemann & Co., whose advertisement appears on page 9 of this issue.

121 East 60th Street.

FRONTAL LOBE ABSCESS SECONDARY TO FRONTAL SINUS INFECTION.*

Dr. Bloyce Hill Britton, El Paso, Tex.

With the present alarming mortality rate for brain abscess, which is estimated upon reliable authority at 75 per cent, more energetic inquiry should be made into the cause of this fact. From the above figures it would seem that the majority of brain abscess diagnoses are made in the postmortem room. With the improved technique of modern surgery this should not be true, and it becomes necessary to seek for more logical reasons.

The most important cause must be attributed to the lack of early diagnosis with subsequent operative interference, rather than late diagnosis, when operative procedure may become only a means of hastening the end. The diagnosis should not be entirely dependent upon neurological findings nor the other frank symptoms which are so consistently absent until the patient is in a crisis. This can be no more aptly expressed than by Dixon[1], who says:

"It is no wonder that we so frequently fail in the early recognition of the vague, yet important symptoms. We diagnose disturbances elsewhere in the body, by deformity, inflammation, pain, fever and impairment of function. All of these may be so slightly disturbed by a brain abscess as to pass unrecognized. I believe we have been led to overrate the value of neurological findings in the early diagnosis of brain abscess and have neglected the most important aid—the history." He further states, "There is no organ in the body that can conceal its trouble so carefully as the brain which harbors an abscess, nor is there a pathological lesion that explodes with such sudden and fatal results."

Abscess formation, particularly in the frontal lobe of the brain, is in a relatively quiescent area of the cerebrum. Involvement of this area may be quite extensive without producing any localizing signs other than perhaps the loss of smell on the affected side. The usual signs of intracranial pressure—headache, vomiting and increase in spinal fluid pressure—are present, but always late in their occurrence. This extensive involvement with no localizing signs must be borne in mind when by a matter of exclusion and history the evidence points

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to the suspicion of a frontal lobe tumor or abscess. Changes of mentality, manner or personality seem to be fairly constant early symptoms. The patient may become irritable, solemn and morose or may have a tendency to witticisms. Abscess of the frontal lobe secondary to frontal sinus infection is not uncommon; yet it is infrequent enough for grave errors to arise, due to not suspecting its occurrence when there is an absolute absence of such local symptoms as tenderness, swelling or pain of the involved area.

Fig. 1. Case 1. Photograph taken three weeks after patient left hospital. Large arrow points to extensive brow incision, through which brain abscess was drained. Small arrow shows exploratory incision into right frontal sinus.

These last-mentioned symptoms and signs, which are so characteristic of the streptococcus mucosus capsulitis, were absent in my patient.

REPORT OF CASE.

P. B., male, age 28 years, was admitted to Hotel Dieu Hospital on March 21, 1931. For 10 days prior to his admission he had been suffering with a severe cold and upper respiratory infection. He had not consulted a physician. Five days prior to his admission his wife noticed a marked change in his personality. He was morose, solemn and irritable; whereas his usual disposition was a cheerful one. Two days prior to his admission he fell from a tractor while plowing in

the field. This apparently did not injure him, but he became dizzy and returned to his home complaining of severe headache and dizziness. He did not at this time consult a physician. The following day he ran a high fever and was more or less delirious, and was taken to the hospital. On admission temperature was 101°F., pulse 78 and respiration 20. He was rational at intervals, during which time he complained bitterly of bitemporal headaches. Examination of the eyes showed the pupils to be equal, of normal size and round, reacting normally. The fundus showed no changes in the nerve head or distension of the retinal vessels. He had had, however, severe photophobia. Examination of the nose showed the mucous membrane to be slightly injected but normal in consistency; many crusts were present in both sides. There was no edema and the nose was well open and ventilated. Nasopharyngoscopic examination showed no

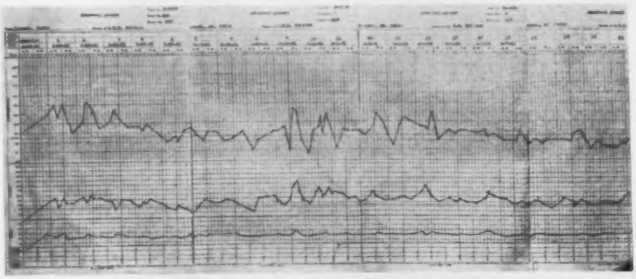

Fig. 2. Temperature record shows elevation of temperature on ninth day, at which time he had an osteomyelitis of the skull.

abnormal findings about the various sinus openings, particularly the frontal. Percussion and pressure over both frontal sinuses elicited no tenderness. Transillumination showed a slight bilateral cloudiness. The throat was slightly red. Otoscopic examination was entirely negative. General examination by an internist revealed nothing more than a slight rigidity of the neck. Neurological examination was entirely negative. Blood examination: Hg., 89 per cent; R. B. C., 4,450,000; W. B. C., 17,300; and differential count: 91 polys.; one large mononuclear and eight small mononuclears. Wassermann, negative. He was put to bed and given morphin to keep him quiet and an ice cap applied to his head. I was called to see him on March 22 at 3:30 A. M. on account of projectile vomiting. Dr. Gorman was called and a spinal puncture was done. Fluid findings: Marked pressure, turbid; (30 c.c. of fluid was withdrawn). Cell count, 1410; 80 polys.; lymphocytes, 56 small and 24 large; 14 polys.; 6 variation.

Globulin increased; sugar normal. No bacteria on smear. Culture was made. X-ray of sinuses: Left frontal cloudy, 3; right frontal cloudy, 2; left antrum cloudy, 3; right antrum cloudy, 2; ethmoid cloudy, 1; sphenoid cloudy, 1. (Scale, 1 to 4.)

March 22, 7:00 P. M.: Temperature 102°F., pulse 90, respiration 20. Patient complained of severe headache and another lumbar puncture was made; 30 c.c. of fluid was removed under marked pressure. It was turbid, with a cell count of 1310. There was the same relative proportion of cells.

Fig. 3. Small arrow points to cloudy left frontal sinus. (The photograph was developed in reverse order, giving the impression of the haziness being in right frontal sinus.)

March 23: Temperature 102°F., pulse 80. Patient complained of headache and vomiting, and was delirious. Puncture made at this time showed the fluid to be under marked pressure, but generally the same findings. Consultation was called relative to the opening of the left frontal sinus, but an agreement was not reached.

March 23, 5:00 P. M.: Report from laboratory this A. M. shows cultures from spinal fluid were negative. Patient was growing progressively worse. Opening of the frontal sinus decided upon, with tentative diagnosis of left frontal lobe brain abscess.

Operation: Incision was made from the lateral extent of the brow margin through the brow over the supraorbital ridge to the base of

the nose. An opening was made with a gouge into the left frontal sinus. Free, tenacious, thick pus resembling cottage cheese was encountered. This was under marked pressure. It was removed with a sponge and curette. A large opening, one-half inch in diameter, was found in the posterior sinus wall just above the cribriform plate. While feeling about this opening with the finger, suddenly I pushed into a large abscess cavity, which was continuous with the opening in the sinus wall. Upon removal of the finger, this same type of cheesy material swelled into the sinus. The lateral walls of the abscess were easily palpated but the posterior wall could not be felt. The bony

Fig. 4. Arrow points to approximate area of frontal lobe brain abscess.

opening was enlarged with rongeur forceps. A large, soft rubber drain was inserted into the abscess cavity. Irrigation through the sinus showed the nasofrontal canal to be open. The wound was partially closed with dermal sutures. The right frontal sinus was opened for exploration and found to contain a small amount of serous fluid. This was drained with a small cigarette drain and closed with dermal sutures. The operation was done under local anesthesia which was very satisfactory (see Fig. 1).

Postoperative Course: Immediately following the operation, the patient slept for a period of eight hours. He did not complain of headache nor did he vomit.

March 24: The day following the operation, he complained of headache, but did not vomit. Spinal puncture was done; fluid was found under slight pressure and was turbid. Cell count at the time was 810, with 18 per cent lymphocytes. The drainage from the tube was a bloody, half-coagulated pus. Temperature 99°F., pulse 86.

March 25: Patient appeared improved, taking fluids and liquid nourishment. However, he complained of headache, and a spinal puncture was done. The pressure was normal, but the turbid fluid had a cell count of 428, 78 of which were lymphocytes.

March 26: For the following four days the patient's temperature ranged from normal to 99.5°F. He was clinically, much improved

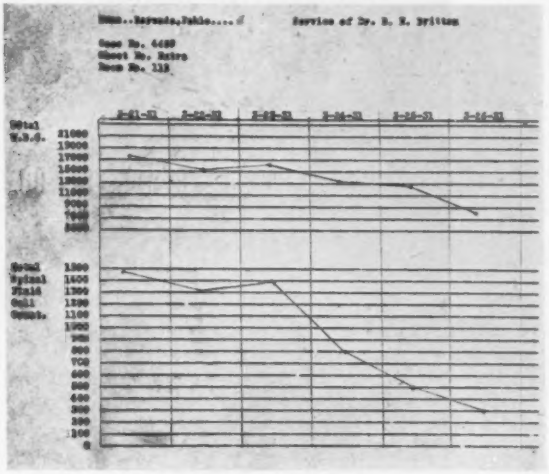

Fig. 5. Graphic chart showing comparative likeness of total white blood count, and spinal fluid cell count.

and was taking nutrition very well. However, on March 30 his temperature suddenly went to 102°F. and he was restless, but did not complain of any pain. Examination of the wound over the left frontal sinus around the tube showed the skin red and angry, with tenderness of the adjacent bone of the skull. The sutures were removed from the wound and the frontal sinus and abscess cavity were irrigated with Carrell-Dakin solution. He apparently had developed some osteomyelitis of the skull adjacent to the operative field. These irrigations were continued for a period of five days, during which time the drainage tube was gradually removed and his temperature

again reached normal, and did not vary from that time until his release from the hospital 21 days following his admission. The wound healed rapidly and there were no other complications (see Fig. 2).

Bacteriology and Etiology: The streptococcus mucosus capsulitis has been more or less commonly found in suppurations of the middle ear and almost invariably associated with mastoiditis and its complications. A characteristic feature of the organism is that it does not produce any of the symptoms of other acute infections, and is generally not recognized until after complications have developed. It has been described by some as being a Type III pneumococcus. It has been found that this organism is not bile soluble, showing it to be a true streptococcus. This patient had an acute frontal sinus infection from a streptococcus mucosus capsulitis, behaving in its most flagrant manner, in that there was no local pain or tenderness, but there was rapid bone necrosis. From the limited literature that I have read on this organism, I can find no explanation for its peculiar affinity for bone, and its apparent impotency in soft tissue. There must be some organic salt contained in bone which is necessary for its rapid growth.

COMMENT.

The nasofrontal duct in this case evidently had become occluded, due to the edema and thick secretion high up in the duct (see Fig. 3). Therefore, there was no nasal discharge or pus visible at the nasal orifice of the duct. Insertion of a probe was tried when the patient first entered the hospital but, due to a marked deflection of the septum and subsequent distortion of the nasal anatomy, it was impossible to enter the frontal sinus in this manner. Bone necrosis occurred in the posterior sinus wall at its thinnest portion. The dura had become adherent and friable and, due to its extreme friability, it ruptured into the abscess while palpating the dehiscence in the posterior wall of the sinus. Had I known of the laboratory findings prior to operation, I would not have operated on this patient, as it was obvious that he had a meningitis. Two cultures taken from the spinal fluid showed a pure growth of streptococcus mucosus capsulitis; one before operation and one following operation, and the material from the abscess cavity also gave a positive culture. This case certainly presented a factor of futility for any operative interference. The only reason the case was operated was due to the fact that the laboratory findings were delayed and the patient was growing progressively worse; confident that it must be a left side frontal lobe abscess, in view of the X-ray findings; and that in this silent area of the brain an abscess could

develop with no localizing signs, the operation was performed. The case is unusual, in that the patient recovered while having a frank streptococcus mucosus meningitis. The explanation for this recovery, I think, is based on: *1*. The low virulency of the organism; *2*. The high resistance of the patient to the organism; and, *3*. Free drainage both from the abscess cavity and from the spinal canal by repeated frequent lumbar punctures (see Fig. 4).

SUMMARY.

1. A case of frontal lobe abscess secondary to frontal sinus involvement is presented.

2. The invading organism was the streptococcus mucosus capsulitis.

3. Prior to operation the symptoms were: Vomiting, headache and a slight rigidity of the neck. There were no localizing symptoms.

4. The frontal sinus was opened and a necrotic dehiscence of the posterior wall was found communicating with an abscess cavity in the left frontal lobe of the brain, measuring approximately, 3x4x6 c.m.m. This abscess was drained.

5. The brow incision under local anesthesia was a satisfactory method of approach.

6. The case is unusual, in that the patient recovered in the presence of repeated positive spinal fluid culture, showing the streptococcus mucosus capsulitis.

7. The terminal meningitis resulting from a ruptured brain abscess may be the only part of the diagnosis which is made in these patients.

REFERENCE.

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502 Two Republics Building.

ZINC IONIZATION IN THE TREATMENT OF CHRONIC SUPPURATIVE OTITIS MEDIA.

DR. N. ASHERSON, London, England.

The purport of this communication is to once more draw attention to a method of curing chronic suppurative otorrhea by means of zinc ionization; to indicate the exact type of case that will respond; to describe the assembling of a suitable apparatus for the purpose, and one that can be easily constructed by anyone.

For any otologist to disregard this method is to confess to ignorance of a treatment that is almost 100 per cent effective, in the suitable case.

The application is so simple that anyone can apply it.

Even in these enlightened days when all knowledge spreads rapidly, it is surprising to find how many practicing otologists are unfamiliar with the use of this method; while many who have not realized the type of case in which to apply it, openly sneer at its use.

The following components are required: *1.* Four or more 2-volt dry cells, size about 1½ by 1½ by 4½ inches; *2.* a sliding or circular rheostat of 2500 ohms resistance, a circular wireless type is applicable; *3.* a milliammeter, with a two-way reading, up to 10 milliamperes; *4.* a switch; *5.* two terminals; *6.* a pair of insulated leads, about 3 feet long each; *7.* a sheet of lead, 3 by 6 inches and 2 to 3 millimetres thick, to which a terminal has been fixed; *8.* a vulcanite aural speculum, to which is attached a zinc rod, running its entire depth, to the narrow end; at the free end a terminal is attached; *9.* a box with a panel, a discarded battery box will be found of use for this purpose.

The circuit is given in the schematic plan. The cells, three in number, are connected to each other in series. The positive pole is connected through the rheostat to the milliammeter, and then to the positive terminal, which should be labeled thus. The negative pole is connected through the switch to the other terminal, which is also labeled. The aural speculum lead comes off the positive pole. The indifferent electrode comes off the negative pole.

The set is arranged so that the rheostat, the milliammeter, the switch and the terminals are affixed on the panel of the box by screws in the manner indicated in the plan.

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The current required is three to five milliamperes, and this is easily obtained from three dry cells (two volts). As a rule for ionizing the ear the current from two such cells will be found to be effective.

The rheostat must be of the sliding adjustment type, and must have a resistance of not less than 2500 ohms. The type of resistance found in the physic laboratories will fill this want, though a rough and effective resistance will be available in the wireless resistances, consisting of a circularly wound coil, of 2500 ohms resistance.

The milliammeter reading from 0 to 10 milliamperes should be of the type with a needle centrally pivoted, so that no matter to what side the leads come, a reading will be available. If it is only a one-way reading, the circuit must be arranged so that when the circuit is completed the needle records the flow through the instrument.

The aural speculum must be of a small size, so as to be available for use with children. It is made of nonconducting material, such as vulcanite, and running down its lumen to the beak of the instrument is a zinc wire, with a terminal at its free end.

The pliable lead sheet, 6 by 3 inches, is for making contact from the negative pole and so completing the circuit through the patient.

Solutions used: Zinc sulphate, gr. 5; glycerin, c.c. 60; aqua ad, 1,000 c.c. For use, dilute with an equal part of warm water.

Before using the apparatus it should be tested by placing the two leads on a piece of lint soaked in saline or zinc solution. The ends are placed one inch apart. The switch is turned on, and the resistance decreased gradually. The ammeter will show if the current is flowing and the amount.

To test which is the positive pole, a piece of moistened litmus paper is placed on the moistened lint, and the point of the electrodes placed on it. The positive pole turns red. The central terminal on the dry cell is the positive pole.

From the treatment point of view it is of the utmost importance to establish the site of the perforation in the tympanic membrane. Thorough cleansing of the meatus is necessary.

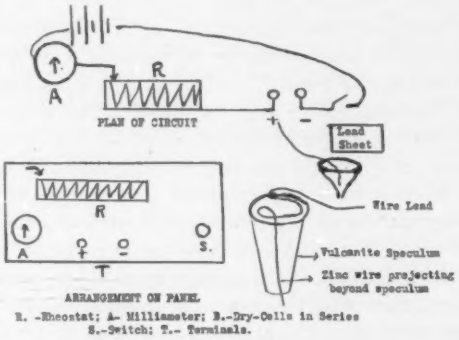
Cases of chronic suppurative otitis media may be divided into two categories: *a*. those with central perforation, and *b*. those with marginal perforation. The latter is invariably associated with granulations, and the common picture in this group is the posterior superior marginal perforation with granulations, and the presence of secretions in the attic (attic suppurative). In passing, one may add that the use of iodin powder, as described by Lederman, in this group, has in many cases proved a curative procedure, though it is not possible to foretell which case will respond, and which will fail.

Cases of marginal perforations are not considered here, and to use ionization for them and expect a cure is to reveal an ignorance of the case in which ionization is indicated. Cholesteatoma and fistula are excluded.

By central perforation is meant that the perforation, however large, does not extend to the margo tympanicus. It is as a rule the external visible physical sign of infection limited to the tympanic lining membrane in chronic cases.

Ninety-five per cent of cases of chronic suppurative otorrhea with a central perforation can be cured with ionization, with certainty.

Mere duration of the discharge is, *per se*, no indication for operative procedures. Only recently I have had a case of chronic otorrhea which was on the verge of being operated upon by a competent



R. -Rheostat; A- Milliameter; B.-Dry-Cells in Series
S.-Switch; T.- Terminals.

aural surgeon of much experience. The case had a central perforation, and was cured of the otorrhea by one treatment with zinc ionization.

Zinc Ionization for Chronic Suppurative Otitis Media. Choice of Suitable Case: The type of suppurative otitis media that responds specifically to ionization is the type coming under the heading of tympanic infection. That is to say, the suppuration is limited solely to the mucous lining of the tympanic cavity. There is adequate drainage present, and no bone suppuration is present, as revealed by the presence of granulations, polypi, etc. The case is not one of cholesteatoma. No complications of the otorrhea are present, *e. g.*, cerebral or mastoid extensions. The perforation is central.

One may cynically retort that the remaining class of case, after eliminating these, is the case, par excellence, that will cease dis-

charging under any treatment. This may be true in part only, but it is certainly not the whole truth.

Two hundred and forty cases out of 340 consecutive cases of chronic otorrhea came under the class of tympanic infection. Many of these cases had been discharging for years and months in spite of active treatment, which failed to cure the condition. Of the 240 cases, in no case had the discharge been present for less than a few months; many had been present for years. Moreover, the naso-pharynx had been attended to without any impression on the otorrhea. Only recently a case of chronic otorrhea presented, following measles many years ago, since when the otorrhea had been persistent, without any remission. There was a central perforation, with no granulations. The ear was cleansed thoroughly, and ionized with zinc sulphate for 10 minutes with three milliamperes. The discharge ceased forthwith, and the ear dried up completely with the single treatment. This history of dramatic cessation of a chronic otorrhea is absolutely typical of the reaction of all such cases to ionization.

If the correct case is treated, the ionization will succeed in almost 95 per cent of cases, with a single application. Only observation is further required in the routine after care. The discharge, present so long, ceases dramatically, and by producing this instant cessation, the treatment by ionization must obtain a front rank in all treatments of chronic suppurative otitis media, due to tympanic infection.

The duration of the discharge is of no importance. No matter how long it has persisted, if the condition is due to tympanic infection, as shown chiefly by the presence of a central perforation in the tympanic membrane, the case can be cured by ionization, and that by a single application in 95 per cent of all such cases. The exceptions will be dealt with later.

Preliminary Treatment: This is important, and must not be skimped. This is directed to the choice of the suitable case (see above), eliminating that due to bone suppuration, the presence of granulations or cholesteatoma.

The ear is thoroughly cleansed by syringing, and mopping with wool applicators. If this cannot be accomplished at one sitting, the ear must be cleaned daily until, pus, crusts sloughs have been removed. It is sometimes difficult to remove the "oyster of mucus" that collects in the depth of the tympanic cavity, and whose presence can be detected by the use of the aspirating speculum (Siegle).

It is assumed that the perforation is sufficiently patent to permit of proper drainage. If it is small or placed high and so obstruct drainage, the perforation must be enlarged before ionization.

After the ear has been cleansed, the tympanic cavity is aspirated by means of Siegle's speculum, to see if there is any secretion pent up in the depth of the tympanic cavity. This if present must be removed.

No ionization must be undertaken while any mucus or pus remains in the tympanic cavity, as its presence prevents the ionization fluid from obtaining contact with the entire tympanic cavity and so a failure may result.

It is obvious that any nasopharyngeal pathology must be attended to as well, *e. g.*, tonsils and adenoids, sinuses, etc. No permanent benefit will accrue if this part is neglected. This by no means betokens delay until this is rectified, but the matter must receive attention if the ear is to remain dry. But ionization can dry an ear in the presence of these sources of reinfection.

If a long series of cases, in which the ear has been rendered dry by ionization, be followed up for years, it will be found that a small but definite return in a few months to a few years, with a recrudescence of the discharge. The reinfection can in these cases be invariably traced to nasopharyngeal infection which has been neglected.

Such number under 5 per cent of all cases. This in no way detracts from the value of the ionization, but emphasizes the necessity of paying attention to the abnormalities of the nose and nasopharynx.

The persistence of the perforation (*i. e.*, its failure to heal) after the ear has become dry, renders the case liable to reinfection.

Method of Application: The ear being cleaned, it is finally syringed with the zinc sulphate solution; this is known as the ionizing fluid. The patient lies on a couch with the ear to be treated uppermost. The negative electrode is firmly bound to the bared wrist or calf, over three layers of lint saturated with a 10 per cent saline solution. The lint must be moist, and the lead sheet must be moulded firmly to the part so as to obtain good contact. The electrode is further kept in position by being firmly bandaged with an ordinary bandage.

The lead is firmly attached to the terminal.

The vulcanite speculum is gently inserted into the meatus so that its axis is almost vertical. It is filled with the ionizing fluid, which thus fills the meatus and tympanic cavity. No air bubbles should be present. These can be eliminated, by first filling the meatus with the fluid, and then pushing the end of the finger into the meatus. This will force out any air bubbles. The speculum should be wedged in firmly so that it can remain *in situ* without further support.

The lead from the speculum is connected up to the positive terminal on the instrument.

To insure good contact of the zinc wire in the speculum with the fluid in the meatus, a loose piece of cotton wool saturated with ionizing solution is pushed down the lumen of the speculum with forceps. This will conduct the current from the wire to the fluid column in the ear.

The switch is closed to complete the circuit, the resistance being a maximum.

The current is gradually increased, slowly by decreasing the resistance, to 3 milliamperes. The latter is reached in 30 to 60 seconds. The ammeter needle must move slowly and gradually. If it oscillates to and fro there is a loose connection in the circuit and this requires rectification.

Some giddiness may be experienced, and is transient. If it is severe, the current must be diminished.

It is maintained at 3 milliamperes for 10 minutes, when the current is very gradually diminished to zero before the circuit is broken by the switch.

If the current has not reached zero, when the switch is opened, there may be a momentary twitch and flash of light experienced by the patient, who may also experience a tingling all the while at the place where the negative electrode is applied. But the method is free from pain or discomfort, and can be used in the youngest of children without fear or frightening them. In fact, children stand the ionization extremely well.

The meatus is dried with cotton wool, and if the ear is examined now, a white eschar will be observed occupying the depth of the meatus.

Boric acid is now insufflated and the patient instructed to attend in a week's time, and not to do anything to the ear in the meanwhile.

The usual sequence is for the discharge to cease completely with the single treatment. The case is seen at weekly intervals for four weeks, after which the case can be discharged from observation. The only treatment is the insufflation of boric acid at each attendance.

In the remaining 5 per cent of cases it will be found that although the otorrhea has diminished in amount, it is still present. This is probably due to the fact that the ear has not been cleaned properly. The ear must now be cleaned and ionized again. In a very small number of cases as many as three or four ionizations at weekly intervals may be necessary to cure the otorrhea. Mucus or pus or

adherent sloughs in the tympanic cavity will prevent access of the ionizing fluid to all parts of the tympanic cavity, and so the ionization is patchy instead of being uniform.

There do occur cases of apparent tympanic infection as shown by the presence of a central perforation, and absence of granulations, in which the ionization proves a failure, even when repeated at intervals, and care has been taken to clean the tympanic cavity thoroughly.

It will be found that the persistence of the condition is due to a polypus deep to the perforation, and strong aspiration with a Siegle speculum will draw it down into view through the perforation.

Three cases out of 240 cases of chronic otorrhea in children with a central perforation showed this underlying pathology.

London, W1, England.

BOOK REVIEW.

Food Allergy. Its Manifestations, Diagnosis and Treatment, with a General Discussion of Bronchial Asthma. By Albert H. Rowe, M.S., M.D., Lecturer in Medicine in the University of California Medical School, San Francisco, Calif.; Chief of Clinic for Allergic Diseases, of the Alameda County Health Center, Oakland, Calif.; Consultant in Allergic and Metabolic Diseases, Highland Hospital; President of the Association for the Study of Allergy, 1927-28. Philadelphia: Lea and Febiger, 1931.

The Rhinolaryngologist, because of his interest in Allergy, must keep abreast of the trend as manifested by those who are delving deeply into the subject.

As a Laryngologist, it strikes the reviewer as a somewhat hopeless task when an authority on the subject may write a text of more than 400 pages on the allergies due to food. Using this as a yard-stick, a complete textbook on allergy would, of necessity, contain more than 1,000 pages. What proportions of this voluminous data can or will be of value to the Rhinologist?

Dr. Rowe has compiled a most thorough volume on the relationship of food to allergy. Food-sensitive tests are carefully outlined, history forms suggested, and treatment prescribed. A separate chapter deals with gastrointestinal food allergy. Bronchial asthma, eczema, urticaria, angioneurotic edema, generalized edema, allergic migraine and neuralgia are all discussed with particular reference to food allergy.

A summary of literature on the subject with a complete bibliography renders this book of value as a reference. M. F.

THE CONTROL OF PAIN IN INTRAORAL CANCER.

Dr. G. Allen Robinson, New York.

A marked inflammatory reaction in the cancerous tumor tissues will appear usually three to five days after the application of radium. Pain may be increased following the treatment and last two to four weeks, or until the reaction has subsided. In intraoral cancer the distress is particularly intolerable when the lesion is in the epiglottis or arytenoid region. Deglutition then becomes a matter of extreme dysphagia and relief assumes the importance of sheer necessity.

Whenever possible, I have endeavored to mitigate the suffering by localized measures, restricting the use of salicylates and opiates to deep-seated lesions. Topical application of cocain is often very effective, but not practical and at times contraindicated. Orthoform powder and similar mild anesthetics may prove of value if the pain is not as severe as it is likely to be in the majority of cases. In other words, there is a need for a local anesthesia, adequately powerful with negligible toxicity, which the patient can administer before meals and whenever the pain becomes severe.

During the past six months I have tested euphagin tablets in a series of twenty-five cases of carcinoma of the tongue, inside of cheek, pharynx, epiglottis, esophagus, larynx and palate, and lymphosarcoma of the tonsil. The active anesthetic principle of euphagin is amidobenzoicethylester. About ten to fifteen minutes before meals the patient will allow one lozenge to dissolve slowly in the mouth. If possible, the table should be placed in contact with the lesion. The ensuing desensitization of the mucous membranes will last about one hour, giving the patient an opportunity to partake of his meal without excessive discomfort.

My experience with euphagin has so far been very gratifying. It appears to possess sufficiently powerful anesthetic properties to mitigate the suffering even in very severe cases. It is conveniently administered and well tolerated, as indicated by the absence of gastric disturbances or other untoward effects. In the Radium Clinic at the Manhattan Eye, Ear and Throat Hospital euphagin is now being dispensed routinely and is considered an effective substitute for narcotic stimulants in conditions indicated for its use.

2 East 77th Street.

International Digest of Current Otolaryngology.

Editor:
Dr. Maxwell Fineberg, St. Louis.
Collaborators:
Prof. G. Bilancioni, Rome.
Mr. W. S. Daggett, London.
Priv. Doz. G. Kelemen, Budapest.
Dr. H. C. Rosenberger, Cleveland.

The Prospectus of the Annual Spring Conference of the Dallas Southern Clinical Society has been published. The dates of the meeting are from March 28 to April 2, 1932. A notable list of speakers has been secured. Otolaryngology forms one of the major subjects of the meeting with numerous symposia scheduled on the subject and its borderline relationships.

The meeting of the American Academy of Ophthalmology and Otolaryngology will be held in Montreal, Sept. 19 to 23. Headquarters will be the Mount Royal Hotel.

The American Board of Otolaryngology will hold an examination, Saturday, Sept. 17, at the Royal Victoria Hospital. Prospective applicants should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for proper application blanks.

The Cleveland Clinic Foundation offers a limited number of two-year Fellowships in Ear, Nose and Throat, which give a varied and practical experience in this subject. Approximately one-half of the time is spent in diagnostic work in the clinic and the other half in hospital service.

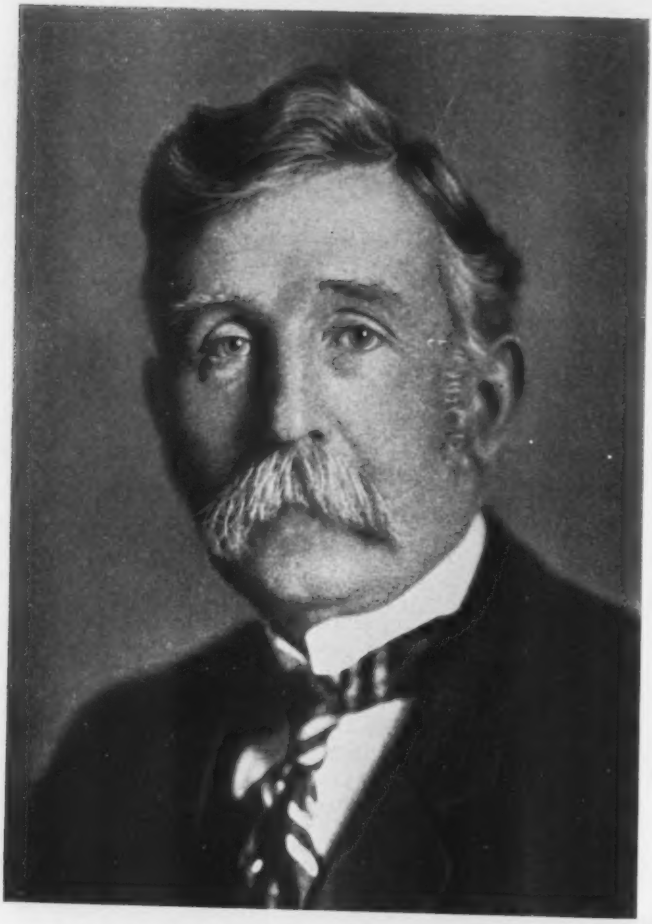
It is desired that the applicants have a fundamental training in the anatomy and physiology of the ear, nose and throat. They will be given a thorough training in history taking, diagnosis, treatment, medical and surgical, and in addition will be given ample opportunity to do special laboratory work and to make original investigations.

These Fellowships carry a payment of $1,000 for the first and $1,250 for the second year. Appointments are effective July 15th of each year.

A course of Fellowship lectures on general subjects extending over two years is given on each Monday evening from September to May, inclusive.

The Fellows are expected also to attend the weekly Pathological Conferences and the regular weekly meetings of the Medical Staff, at which various clinical problems are discussed.

Application blanks will be forwarded upon request to the Secretary of the Fellowship Committee, Cleveland Clinic, Cleveland, Ohio.



BURTON ALEXANDER RANDALL.

IN MEMORIAM

BURTON ALEXANDER RANDALL.

Dr. B. Alexander Randall, Emeritus Professor of Otology of the University of Pennsylvania, died at his home in Philadelphia on Jan. 4, 1932, after a long and distinguished service. He received his medical and Ph.D. degrees from the University of Pennsylvania, and M.A. degree from St. Johns. His wife, a sister, two brothers, two sons and a daughter survive him.

Alhough he had retired from active practice, he was prominent in all the otolaryngologic councils, a beloved and respected member of local and national organizations. Commencing as student demonstrator of anatomy in 1879, he was in continuous hospital service until his retirement from the University in 1924, and beside the University his Philadelphia hospital connections had included the Episcopal, Polyclinic, Children's, Methodist, Women's and St. Timothy's.

He was assistant demonstrator of Histology in the University Medical Department from 1880 to 1882, Eye and Ear Surgeon to the Episcopal Hospital from 1882 to 1891, appointed to the Children's Hospital in 1885, Professor of Diseases of the Ear at the Philadelphia Polyclinic from 1888 to 1902, Clinical Professor of Diseases of the Ear, University of Pennsylvania, 1891 to 1912; Professor of Otology, University of Pennsylvania, 1912 to 1924.

Dr. Randall was a Fellow of the College of Physicians of Philadelphia, Fellow of the American Ophthalmological Society, Fellow and former President of the American Otological Society, Fellow of the American Laryngological Association, member of the Philadelphia Pathological Society.

He was an active member and one time President of the A.M.P.O. Medical Fraternity, was active in many civic organizations, and these 44 recorded years of active service left a profound impress in Philadelphia. Somewhat conservative in his later years, his intellectual keenness, inexhaustible memory and rare medical judgment continued until his death. He was a man of rare character, an artist of no mean ability, of a Chesterfieldian grace and culture. His intimate friends and medical associates will never forget his genial, sympathetic nature, generous response to every appeal and devotion to the highest ideals of his profession.

The publications of Dr. B. Alexander Randall were many—perhaps he will be particularly remembered for his co-authorship with Morse of "Photographic Illustrations of the Anatomy of the Ear," and with de Schweinitz of "American Text Book of Diseases of Eye, Ear, Nose and Throat."

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JOHN EDMUND MACKENTY.

Dr. John Edmund Mackenty died in New York on Dec. 11, 1931, of angina pectoris, aged 62 years. His wife, two children and four grandchildren survive him.

He was born in Richmond, P. Q., Canada, and received his preliminary education at Sherbrooke and Ottawa. He graduated from McGill University Medical School in 1892 and came to the United States during the same year. He spent a few years at the Massachusetts General Hospital, Boston, under A. T. Cabot, and then undertook post-graduate work in Europe, in 1899.

For more than thirty years Dr. Mackenty practiced otolaryngology in the City of New York and held numerous university and hospital positions. He was a Fellow of the American Laryngological, Rhinological and Otological Society, the American Laryngological Association, and the American College of Surgeons; he was a member of the New York Laryngological Society and of the New York Academy of Medicine. He spent many years in various positions at the Manhattan Eye, Ear and Throat Hospital and at the time of his death was in charge of one Service as well as a member of the Board of Directors of the hospital.

Dr. Mackenty was a pioneer in laryngeal surgery and had earned for himself an enviable international reputation in this field. For many years he wrote and lectured about the early diagnosis of carcinoma of the larynx and its treatment; he described his technique of operation, and today the Mackenty Method of laryngectomy is a by-word among laryngologists.

As a colleague, teacher, and man he was loved and respected by all those connected with him; his opinions were sought after, and his suggestions were usually accepted with gratitude by his consultants.

Laryngology has suffered a great loss in the death of John Edmund Mackenty. His place as a leader in American laryngology will be hard to fill, and his memory will long be cherished by all who knew him.

Dr. Mackenty was a prolific writer on his subject and frequently pleaded with American readers to help in the early diagnosis of malignancies of the larynx. His last publication, which will appear shortly, is a thorough review of "The Relationship of the Upper Respiratory Tract to Eye Conditions." A full list of his published articles follows:

THE PUBLICATIONS OF JOHN E. MACKENTY.

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THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

Meeting of May 28, 1931.

Studies in Sinus Infection. Dr. J. Wynn Pugh.

(Published in full in THE LARYNGOSCOPE, *October, 1931.)*

DISCUSSION.

DR. H. H. FORBES: It is interesting at all times to have the subject of sinus disease presented to the Section. I do not know of anything more trying in our practice. As for the comparison of the prevalence of cases in this country and on the other side, I remember that in the clinics of Glasgow and Edinburgh there seemed to be as many such cases as we have here, notwithstanding the absence of central heating. However, we do know that there is a certain problem among children, the fall weather filling the clinics. The work in cultivating the bacteria from the sinus washing is most interesting to me and I hope that Dr. Pugh will follow them along and see if we can in some way improve our method of treating these troublesome cases. The angina type of bacteria is interesting and the reaction of the fluid in the sinuses is also interesting, as well as the point he brings out in the use of an alkali in the treatment. I don't know of any other condition of the nose in which so many forms of irrigation have been used. I feel that we ought not to place too much stress on the strong irrigating fluids that some of us have been using. I thank the Doctor for bringing up this subject and hope he will continue his investigations.

I would like to know his opinion as to the strength of the irrigating solutions he has used. I suppose that all the irrigations have been made by puncture under the inferior turbinate bone. I have been irrigating my cases through the natural opening. I don't see why this method could not be used in the investigations. I have found it comparatively easy to find the natural opening, and without pain. So far I have not had a case in which I have failed to irrigate the antrum through the natural opening.

DR. J. W. BABCOCK: I would like to comment on Dr. Pugh's paper in regard to washing out through the normal opening. I have tried that considerably myself, and find a certain percentage of cases where I cannot enter it. It is not accessible in some instances and even when found it does not always give as satisfactory irrigation as the other. I am stating this advisedly, for I have irrigated through the normal opening and followed this by puncture and obtained secretion by the latter procedure. In many instances it is an advisable procedure, but not in all. Dr. Pugh has done some very good work in investigating the sinuses.

DR. MARVIN JONES: In a discussion like this on the sinuses, I would like to bring up the angle of allergic origin in the sinuses. I would like to know how much that has possibly entered into the investigation.

DR. C. J. IMPERATORI: Before the American Laryngological Association at its next meeting, Dr. Mosher, of Boston, will report some investigations that show in a number of patients with chronic maxillary sinusitis the tissues removed had living cilia. Dr. Loré's contention regarding the ciliated epithelium lining the sinuses is very pertinent.

DR. D. J. MCDONALD: We must all admire the work of Dr. Pugh and its presentation in such an excellent paper this evening. Much or little, the economic law of the causes that lead to sinus trouble, particularly in most of our eastern states, has been brought to our attention by the United States Government only within the past few months in regard to the saturation of the atmosphere, seen by the excessive carbon (40 per cent) in New York, due to new apparatus heating, smoke contamination and soft coal. There is not a man here who cleans out the nose who does not find carbon, etc. Dr. Loré has given us a wonderful point, and I have a number of cases in which that line of treatment is being carried out.

I would say to my good friend here that I cannot always get into the sinus: I have used the ordinary probe and have many times been disappointed. Some years ago (June, 1923) before the seventy-fourth meeting of the American Medical Association. I showed the perfected nasal pharyngoscope. This instrument should be used in many cases as it brings out many salient points. Dr. Charles Richardson ordered 24 of these instruments, and they are now being used extensively in the United States Army. I have seen the improved nasal pharyngoscope used in London, also in Hajek's Clinic, and the improved apparatus for examining the sinus.

I feel that the Health Division of the Academy, with the aid of the Health Department of the City of New York, ought to stop this smoke nuisance. In my particular neighborhood several institutions, and others that I have been connected with, have done much to prevent it. In the schools we found on investigation that 8 to 10 per cent of the school children had sinus involvement. If the gentlemen who have done so much tonight in bringing out the bacteriological facts will continue this work, and also Dr. Loré, I think we may hope for much better results in these cases.

DR. PUGH (closing): Perhaps I ought to explain that my paper was in no way intended to be comprehensive, but merely presented that part of the field in which I have been working; that is, the bacteriology of these infections. I seem to have raised several waves of dissension and discussion about the method of irrigation of the maxillary sinuses. I had not intended doing so, but I realize it is a matter of importance.

In reply to a doctor (whose name I did not catch), who had spoken enthusiastically in favor of irrigating the antra exclusively through the natural ostium: In this matter of irrigation it seems to me mechanically unsound to wash out any chamber through a single small orifice which is placed near the top of the said chamber, whether with or without the use of compressed air. The infected sinus may be regarded in many ways as a septic tank, and unless one removes the sludge from the bottom of the cavity one cannot claim to have irrigated the sinus in a proper surgical manner. Irrigation through a puncture beneath the inferior turbinate is not painful if a proper local anesthesia and technique is used; and I find it more satisfactory than to attempt an irrigation which even under the best conditions cannot give the same free irrigation of an infected cavity.

Replying to Dr. Forbes: The lotion I have found most satisfactory is an alkaline wash with a pH of 8.0 to 8.2.

Dr. Loré has given me great assistance. His suggestion of Ringer's solution is very sound, and I shall take great pleasure in trying out his advice.

Foreign Bodies in Air Passages. Dr. Geo. R. Brighton.

(Published in full in THE LARYNGOSCOPE, *October, 1931.)*

DISCUSSION.

DR. CHARLES J. IMPERATORI: Dr. Brighton is to be congratulated on the very skillful way in which he removed the foreign body. Unfortunately, he missed one very important point; that is, to insist that all children be rayed from the occiput to the tuber ischi.

DR. H. H. FORBES: Someone told me recently, in reference to the subject of children swallowing safety pins, that children are great mimics, and that as the average mother in changing the baby's diaper generally holds the pin in her mouth, the baby sees this and is apt to repeat the same performance. This may be a reason for trying to get a little more information to mothers in the handling of safety pins. A suggestion was made some time ago in regards to mothers forming a closed safety pin society—keeping the pins closed until it is time for them to be used.

Observations on Paralysis of the Recurrent Laryngeal Nerve. Dr. Alfred Michaelis.

(Published in full in THE LARYNGOSCOPE, *October, 1931.)*

Agranulocytic Angina. Dr. Jas. W. Babcock.

(Published in full in THE LARYNGOSCOPE, *November, 1931.)*

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

POSTGRADUATE SESSION.

Regular Meeting of May 5, 1931.

(Continued from Page 80, January, 1932.)

I am glad that you called attention to the blood stream. It is possible to have maxillary infection through the blood stream on account of its anatomical relationship.

DR. STOUT: If you inject lipiodol in one antrum only and use that as a diagnosis you may make an error. It is best to wait about a week and do the other side, then compare the two sides. You will see curious shapes to antra that are not pathological.

Concerning foreign bodies in the antrum, sometimes small rubber tubes used for drainage will slip into the antrum but if you just give it a little time, Nature will sometimes bring the tube out through the normal ostium into the nose. I have seen this occur twice. One case was a physician's wife.

I will just mention one or two cases to show that you have to be thinking about antrum trouble all the time. Some time ago a patient was sent to me by a dentist. He had extracted a tooth for pain in the jaw and the pain continued afterwards. This patient was given hypodermics of morphia and then several quarts of whiskey to relieve the pain. This was given him by a physician. On his own account he took four or five bottles of Atwood's Bitters. On washing his antrum, a thick, foul mass of pus came out. Shortly after this he developed delirium tremens. The pain was relieved after the washings and it required about a half-dozen washings for complete recovery.

I will cite another case; a woman sent as a possible allergic case. She gave a history of having had a tooth extracted and from that time on had great discomfort. I washed out her antrum and got the usual amount of pus. Another curious thing was that after it was all over I looked into her nose and the swelling had gone down. The whole picture had changed. Her allergic symptoms disappeared at once.

DR. WM. ERSNER: I wish to add as well as to emphasize to this most valuable discussion the fact that there must be whole-hearted and unstinted co-operation between the rhinologist and the dentist or oral surgeon. The results are always more gratifying, with final benefit to the patient.

The rhinologist can be of immeasurable value to the dentist when he needs him. He will be called by the dentist, who has forced a root or tooth into the antrum, asking for instructions. I usually advise placing a small piece of iodoform gauze over the socket (not in the antrum), and with brass ligature wire, figure eight about the adjacent teeth, to which the gauze is tied. One of our Caldwell-Luc cases, a physician, had iodoform packing in the sinus for over two years. In a series of 150 maxillary operations, as read before the college, we found 25 per cent were dental in origin. Perhaps our percentage is higher than usual. This is due to our close co-operation. The majority of these cases were referred directly to me by the dentist.

Our experience leads us to conclude without any hesitation that the sinus should be drained and washed through the nose. Nature intended it to be that way. I do not irrigate through the socket. The mouth opening is immediately closed, before cicatricial tissue forms. If this should happen, we must resort to plastic closures. The sinus is then irrigated by the laryngologist, my duty being too stand by, caring for the oral phase. Invariably the condition clears up and the oral opening closes in a very short time. If the sinus is washed and irrigated through the oral opening (tooth socket), the treatment is prolonged and many of these openings never close.

During some postgraduate work in one of the New York Colleges, I found the following creed set down: The maxillary sinus was "no man's land." If

the sinus was dental in origin, it was treated and irrigated through the socket. We had a large group of chronic maxillary sinus cases, who reported two or three times a week for irrigation. I do not recall any of these cases clearing up, and there were permanent openings in all, for insertion of the cannula.

In all of our Caldwell-Luc, the Denker or usual mouth closures, followed by irrigation, we have no records of any with permanent openings.

In reference to removal of part of the floor of the sinus or the buccal wall, I might add as in one case extreme force was used with the elevator to remove the upper first molar, the three molars came away with the tuberosity and part of the buccal wall. I might recall several similar occurrences, two of which resulted in malpractice suits. We must bear in mind lues and tuberculosis in these cases.

DR. ALFARO (in closing): Two of the discussors have brought out the fact which I have tried to bring out in the paper, that it is very dangerous to irrigate and pack an alveolar opening and endeavor to effect a cure that way.

In regard to transillumination and soft tissue changes, I do not know, Dr. Macfarlan. It is possible, at least one man told me so, to get a shadow by transillumination when you have a dead tooth without any pain. That particular man believes that the tooth should come out. With certain crowned teeth, the metal seems to give a certain amount of darkness to transillumination and it seems to give you a suspicion of alveolar involvement when there is none.

Dr. Ersner reports that 25 per cent of the maxillary diseases necessitate surgery. That figure seems high, but when you are doing work in one field you naturally get a greater percentage of the cases. The fact remains that 25 per cent, and perhaps a little more than that, require radical surgery.

Dr. Stout spoke about the dental tubes coming out through the normal fossa. He also spoke about the changes that take place in the shape of the antrum. I have noticed how curiously the shape of the antrum does change.

The Effect of Radical Mastoidectomy On the Hearing Tests. Dr. Joel J. Pressman.

(Published in full in THE LARYNGOSCOPE, *December, 1931.)*

DISCUSSION.

DR. COATES: I wish we could have heard all about Dr. Pressman's paper, but I suppose the hour is too late to go into the whole thing.

It has always seemed to me that you should have at least as good hearing after a radical mastoidectomy as you started with, or even better. It all depends, I think, on the proper care of the cavity. If you allow your ear to fill up with granulation tissue you cannot expect the ear to be better than before. If you have a lot of scar tissue you cannot expect any improvement in hearing, but if you have a nice thin epidermis, you can get results.

A careful study of a high series of these cases would be of very great interest.